
MEDICO-CHIRURGICAL SOCIETY
EDINBURGH

# THE TRANSACTIONS

OF THE

# MEDICO-CHIRURGICAL SOCIETY

# OF EDINBURGH

VOL. XVIII.—NEW SERIES

*SESSION 1898-99*



EDINBURGH: WILLIAM F. CLAY
*PUBLISHER TO THE SOCIETY*
1899

# PREFACE

THE present Volume is the *Eighteenth* of the *New Series*, and contains a record of the work done during the past Session.

That work, as hitherto, embraces the communication of Original Papers; the exhibition of Patients, illustrating rare and interesting forms of disease; and the exhibition of Pathological and other Specimens, so essential to the proper understanding of the morbid changes which take place in the human body.

During the past Session Extra Meetings were held for the exhibition of Patients, Pathological Specimens, Instruments, etc. It is hoped that such Meetings will materially increase the usefulness of the Society.

It is believed that the publication of the Transactions in this permanent form will prove a valuable contribution to medical literature, will encourage the Members to take a more active part in the work of the Society, and will tend in no small degree to increase the influence and usefulness of the Medico-Chirurgical Society of Edinburgh.

WILLIAM CRAIG,
*Editor.*

*October* 1899.

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| | A. Cowan Guthrie, M.B., C.M., *Leith*, | 1890 |
| | Alexander Lockhart Gillespie, M.D., F.R.C.P. Ed., | 1891 |
| 145 | Stewart Stirling, M.D., F.R.C.S. Ed., | 1891 |
| | Francis Darby Boyd, M.D., F.R.C.P. Ed., | 1891 |
| | John Macpherson, M.D., F.R.C.P. Ed., | 1891 |
| | James Smith, M.B., C.M., | 1891 |
| | Norman Purvis Walker, M.D., F.R.C.P. Ed., | 1891 |
| 150 | Alexander Miles, M.D., F.R.C.S. Ed., | 1892 |
| | Robert Abernethy, M.D., F.R.C.P. Ed., | 1892 |
| | Arthur Logan Turner, M.D., F.R.C.S. Ed., | 1892 |
| | G. Matheson Cullen, M.D., C.M., | 1892 |
| | William George Aitchison Robertson, M.D., F.R.C.P. Ed., | 1892 |
| 155 | David Middleton Greig, M.B., C.M., F.R.C.S. Ed., *Dundee*, | 1892 |
| | John Stevens, M.D., F.R.C.P. Ed., | 1892 |
| | James Crawfurd Dunlop, M.D., F.R.C.P. Ed., M.R.C.S. Eng., | 1892 |
| | William Elder, M.D., F.R.C.P. Ed., *Leith*, | 1892 |
| | Robert Stewart, M.B., C.M., | 1892 |
| 160 | William Crawford M'Ewan, M.D., C.M., *Prestonpans*, | 1892 |
| | George Robertson Wilson, M.D., C.M., *Polton*, | 1892 |
| | Richard J. Erskine Young, M.D., C.M., | 1892 |
| | Richard James Arthur Berry, M.D., F.R.C.S. Ed., | 1893 |
| | John MacRae, M.D., C.M., *Murrayfield*, | 1893 |
| 165 | James Harvey, M.D., C.M., | 1893 |
| | Henry Anderson Peddie, M.B., C.M., | 1893 |
| | John Wheeler Dowden, M.B., F.R.C.S. Ed., | 1893 |
| | Alexander Bruce Giles, M.D., C.M., | 1893 |
| | William Basil Orr, M.D., C.M., | 1893 |
| 170 | James Aitken Clark, M.B., C.M., | 1893 |
| | Robert Mackenzie, M.D., C.M., | 1893 |
| | William Ford Robertson, M.D., C.M., | 1893 |
| | James Mowat, M.B., C.M., | 1893 |
| | Charles Arthur Sturrock, M.B., F.R.C.S. Ed., *Dunfermline*, | 1894 |
| 175 | Claude B. Ker, M.D., C.M., | 1894 |
| | John Cumming, F.R.C.S. Ed., L.R.C.P. & S. Ed., | 1894 |
| | Charles E. Douglas, M.D., C.M., *Cupar-Fife*, | 1894 |
| | William Fraser Wright, M.B., C.M., *Leith*, | 1894 |
| | Douglas Chalmers Watson, M.B., C.M., | 1894 |
| 180 | Thomas Easton, M.D., C.M., | 1894 |
| | Archibald Stodart-Walker, M.B., F.R.C.P. Ed., | 1894 |
| | James Cameron, M.D., C.M., | 1895 |
| | James Middlemass, M.D., F.R.C.P. Ed., | 1895 |
| | Lewis Campbell Bruce, M.D., C.M., *Murthly* | 1895 |
| 185 | Robert Miller Ronaldson, M.D., C.M., M.R.C.S. Eng., | 1895 |
| | James Scott, M.D., C.M., | 1895 |
| | John Hardie, M.B., F.R.C.S. Ed., *Stogursey*, | 1895 |
| | John Orr, M.D., F.R.C.P. Ed., | 1895 |
| | James Gibson Cattanach, M.B., F.R.C.P. Ed., | 1895 |
| 190 | James Stewart Fowler, M.D., F.R.C.P. Ed., | 1895 |
| | Ernest George Salt, L.R.C.P. & S. Ed., | 1895 |
| | John Tod, M.B., C.M., *Leith*, | 1895 |
| | William Henry Miller, M.D., F.R.C.P. Ed., | 1895 |
| | Robert Wilberforce Inkster, M.D., C.M., | 1895 |
| 195 | William Leslie Mackenzie, M.D., C.M., | 1895 |
| | John Adamson Honey Duncan, M.D., C.M., | 1895 |
| | William Alfred Johnstone Alexander, M.B., C.M., | 1896 |
| | John Cormack Smith, M.B., C.M., | 1896 |
| | Harry Rainy, M.D., F.R.C.P. Ed., | 1896 |
| 200 | William Alexander Mackintosh, M.B., C.M., *Stirling*, | 1896 |
| | John Anderson, M.B., C.M., *Pitlochry*, | 1896 |
| | Frederick John Turnbull, L.R.P. & S. Ed., | 1896 |
| | George Elder, M.D., F.R.C.P. Ed., | 1896 |
| | Archibald Adam Scot Skirving, M.B., F.R.C.S. Ed., M.R.C.S. Eng., | 1897 |

| | | Date of Admission. |
|---|---|---|
| 205 | Theodore Shennan, M.D., F.R.C.S. Ed., | 1897 |
| | John Macmillan, D.Sc., M.D., F.R.C.P. Ed., | 1897 |
| | John Frank Crombie, M.B., C.M., *North Berwick*, | 1897 |
| | William Arthur M'Cutchan, L.R.C.P. & S. Ed., *Burghill*, | 1897 |
| | David James Graham, M.D., C.M., | 1897 |
| 210 | Robert John Johnston, M.B., C.M., | 1897 |
| | George Denholm Darlington, M.B., C.M., | 1898 |
| | Alexander Cruickshank Ainslie, M.B., C.M., | 1898 |
| | Thomas Jackson Thyne, M.B., F.R.C.P. Ed., | 1898 |
| | James Taylor Grant, M.D., C.M., | 1898 |
| 215 | Neisch Park Watt, M.B., C.M., | 1898 |
| | Frederick William Reid Mackay, M.D., F.R.C.S. Ed., | 1899 |
| | Thomas Paul Gray, M.D., C.M., | 1899 |
| | Robert Black Purves, M.B., F.R.C.S. Ed., | 1899 |

## (*b.*) Members Exempted under Rule V. from paying the Annual Subscription.

| | | |
|---|---|---|
| ** | Andrew Halliday Douglas, M.D., F.R.C.P. Ed., *Peebles*, | 1842 |
| 220 | W. Judson Van Someren, M.D., L.R.C.S. Ed., *London*, | 1845 |
| | William H. Lowe, M.D., F.R.C.P. Ed., *Wimbledon*, | 1845 |
| | George Skene Keith, M.D., LL.D., F.R.C.P. Ed., *Currie*, | 1845 |
| | His Excellency Robert H. Gunning, M.D., LL.D., *London*, | 1846 |
| | Archibald Hall, M.D., *Montreal*, | 1853 |
| 225 | Sir W. Overend Priestley, M.D., M.P., LL.D., F.R.C.P. Ed., *London*, | 1854 |
| | Horatio Robinson Storer, M.D., *Newport, Rhode Island, U.S.*, | 1855 |
| | Thomas Skinner, M.D., L.R.C.S. Ed., *London*, | 1856 |
| | Professor William Smoult Playfair, M.D., LL.D., F.R.C.P.L. *London*, | 1857 |
| | J. Ivor Murray, M.D., F.R.C.S. Ed., *Scarboro'*, | 1857 |
| 230 | Andrew Scott Myrtle, M.D., L.R.C.S. Ed., *Harrogate*, | 1859 |
| | Francis Robinson Macdonald, M.D., *Inverary*, | 1860 |
| | Professor John Young, M.D., *University of Glasgow*, | 1860 |
| | George Thin, M.D., L.R.C.S. Ed., *London*, | 1861 |
| | Professor William Stephenson, M.D., F.R.C.S. Ed., *Aberdeen University*, | 1861 |
| 235 | J. S. Beveridge, M.R.C.P. Lond., F.R.C.S. Ed., *Foulsham*, | 1861 |
| | David Yellowlees, M.D., LL.D., F.F.P. & S. Glasg., *Glasgow*, | 1861 |
| | Prof. Arthur Gamgee, M.D., F.R.C.P. Ed., F.R.S., *Switzerland*, | 1863 |
| | Professor John Cleland, M.D., LL.D., *The University, Glasgow*, | 1864 |
| | Sir R. B. Finlay, M.D., Q.C., M.P., *Middle Temple, London*, | 1864 |
| 240 | Stanley Lewis Haynes, M.D., M.R.C.S. Eng., *Malvern*, | 1864 |
| | James Watt Black, M.D., F.R.C.P.L., *London*, | 1865 |
| | David Brodie, M.D., *Canterbury*, | 1865 |
| | Robert Shand Turner, M.D., C.M., *Keith*, | 1867 |
| | Peter Maury Deas, M.B., L.R.C.S. Ed., *Exeter*, | 1868 |
| 245 | Professor J. G. M'Kendrick, M.D., LL.D., F.R.C.P. Ed., *University, Glasgow*, | 1870 |
| * | Archibald Dickson, M.D., F.R.C.S. Ed., of *Hartree and Kilbucho*, | 1871 |
| | James Johnston, M.D., F.R.C.S. Ed., *London*, | 1871 |
| | J. William Eastwood, M.D., M.R.C.P.L., *Darlington*, | 1871 |
| | Charles A. E. Sheaf, F.R.C.S. Ed., *Queensland*, | 1871 |
| 250 * | Alexander Ballantyne, M.D., F.R.C.P. Ed., *Dalkeith*, | 1871 |
| | Professor J. Bell Pettigrew, M.D., LL.D., F.R.C.P. Ed., *University of St Andrews*, | 1873 |
| * | Andrew Balfour, M.D., C.M., *Portobello*, | 1874 |
| * | Robert Lucas, M.D., F.R.C.P. Ed., *Dalkeith*, | 1875 |

| | | Date of Admission. |
|---|---|---|
| | John Aymers Macdougall, M.D., F.R.C.S. Ed., *France*, . | 1875 |
| 255 | Thomas John Maclagan, M.D., M.R.C.P.L., *London*, . | 1875 |
| | Dr Groesbeck, *Cincinnati*, . . . . . | 1875 |
| | * John Connel, M.D., F.R.C.P. Ed., *Peebles*, . . | 1876 |
| | Professor David James Hamilton, M.B., F.R.C.S. Ed., *Aberdeen University*, . . . . . | 1876 |
| | James Stitt Thomson, M.D., F.R.C.P. Ed., F.R.C.S. Ed., *Lincoln*, . . . . . | 1877 |
| 260 | George Herbert Bentley, L.R.C.P. & S. Ed., *Kirkliston*, . | 1877 |
| | J. Moolman, M.B., C.M., *Cape of Good Hope*, . . | 1877 |
| | Robert Somerville, M.D., F.R.C.S. Ed., *Galashiels*, . . | 1877 |
| | Graham Steell, M.D., F.R.C.P.L., *Manchester*, . . | 1877 |
| | John Brown, M.D., F.R.C.S. Eng., *Burnley*, . . | 1878 |
| 265 | James Allan Philip, M.D., *Boulogne-Sur-Mer*, . . | 1878 |
| | Alex. Robert Coldstream, M.D., F.R.C.S. Ed., *Florence*, . | 1878 |
| | Professor Johnson Symington, M.D., F.R.C.S. Ed., M.R.C.S. Eng., *Belfast*, . . . . . | 1878 |
| | * William Barrie Dow, M.D., F.R.C.S. Ed., *Dunfermline*, . | 1879 |
| | A. D. Leith Napier, M.D., M.R.C.P.L., *Australia*, . | 1879 |
| 270 | Keith Norman Macdonald, M.D., F.R.C.P. Ed., . . | 1880 |
| | John Home-Hay, M.D., M.R.C.S. Eng., *Alloa*, . . | 1880 |
| | * John Hutton Balfour, M.B., C.M., *Portobello*, . . | 1881 |
| | John Mackay, M.D., L.R.C.S. Ed., *Aberfeldy*. . . | 1881 |
| | William Badger. M.B., C.M., *Penicuik*. . . . | 1882 |
| 275 | * Alexander Matthew, F.R.C.S. Ed., *Corstorphine*, . . | 1882 |
| | John Archibald, M.D., F.R.C.S. Ed., *London*, . . | 1882 |
| | James Rutherford Morison, M.B., F.R.C.S. Ed., *Newcastle*, | 1882 |
| | Roderick Maclaren, M.D., *Carlisle*, . . . . | 1882 |
| | J. Maxwell Ross, M.B., F.R.C.S. Ed., *Dumfries*, . . | 1882 |
| 280 | John Carlyle Johnstone, M.D., C.M., *Melrose*, . . | 1882 |
| | F. W. Dyce Fraser, M.D., F.R.C.P. Ed., *Teddington*, . | 1883 |
| | Edwin Baily, M.B., C.M., *Oban*, . . . . | 1883 |
| | William Hy. Shirreff, M.B., C.M., *Melbourne*, . . | 1883 |
| | John Haddon, M.D., C.M., *Denholm*, . . . | 1883 |
| 285 | A. W. Hare, M.B., F.R.C.S. Ed., M.R.C.S. Eng., *Roche*, . | 1883 |
| | Professor G. Sims Woodhead, M.D., F.R.C.P. Ed., *Cambridge*, | 1883 |
| | John Macdonald Brown, M.B., F.R.C.S. Eng. & Ed., *London*, | 1883 |
| | T. Goodall Nasmyth, M.D., D.Sc., *Cupar-Fife*. . . | 1884 |
| | Thomas R. Scott, M.D., C.M., *Musselburgh*, . . | 1884 |
| 290 | F. A. Saunders, F.R.C.S. Ed., *Grahamstown, South Africa*, | 1884 |
| | Joseph Carne Ross, M.D., F.R.C.P. Ed., *Withington*, . | 1884 |
| | G. J. H. Bell, M.B., C.M., *Bengal Army*, . . . | 1884 |
| | W. C. Greig, M.D., C.M., *Morocco*, . . . . | 1884 |
| | J. Craig Balfour, L.R.C.P. & S. Ed., . . . | 1884 |
| 295 | T. Wyld Pairman, L.R.C.P. & S. Ed., *New Zealand*, . | 1884 |
| | * Alexander Thom, M.D., C.M., *Crieff*, . . . | 1884 |
| | Andrew Brown, M.D., M.R.C.P. Ed., *London*, . . | 1884 |
| | Ernest F. Neve, M.D., F.R.C.S. Ed., M.R.C.S. Eng., *India*, | 1884 |
| | James Robertson Crease. F.R.C.S. Ed., *South Shields*, . | 1885 |
| 300 | T. Edgar Underhill, M.D., F.R.C.S. Ed., *Burnt Green*, . | 1885 |
| | S. Hale Puckle, M.B., C.M., *Bishop's Castle*, . . | 1885 |
| | Skene Keith, M.B., F.R.C.S. Ed., *London*, . . | 1885 |
| | Reginald Ernest Horsley, M.D., F.R.C.S. Ed., *Stoneyhurst*, | 1886 |
| | Professor Robert Fraser Calder Leith, M.B., F.R.C.P. Ed., *Birmingham*, . . . . . | 1886 |
| 305 | John Batty Tuke, jr., M.D., F.R.C.P. Ed., *Murrayfield*. . | 1886 |
| | Oswald Gillespie Wood, M.D., F.R.C.S. Ed., Surgeon, *A. M. Staff, India*. . . . . | 1886 |
| | James Hogarth Pringle, M.B., C.M., *Glasgow*, . . | 1886 |
| | Walter Scott Lang, M.D., F.R.C.S. Ed., . . . | 1886 |
| | William Gayton, M.D., M.R.C.S. Eng., *London*. . . | 1886 |
| 310 | A. Bell Whitton, M.B., C.M., *Aberchirder*. . . . | 1886 |

| | | Date of Admission. |
|---|---|---|
| | J. Walton Hamp, L.F.P. & S. Glasg., L.S.A. Lond., *Wolverhampton*, | 1887 |
| | John Keay, M.B., F.R.C.P. Ed., *Inverness*, | 1887 |
| | John F. Sturrock, M.B., C.M., *Broughty Ferry*, | 1887 |
| | David W. Aitken, M.B., C.M., *London*, | 1887 |
| 315 | William Hunter, M.D., M.R.C.S. Eng., M.R.C.P.L., *London*, | 1887 |
| | Sydney Rumboll, L.R.C.P. Ed., F.R.C.S. Ed., *Leeds*, | 1887 |
| | George Franklin Shiels, M.D., F.R.C.S. Ed., *San Francisco*, | 1887 |
| | D. H. Anderson, M.D., C.M., *Barrow-in-Furness*, | 1887 |
| | J. A. Armitage, M.D., C.M., *Wolverhampton*, | 1887 |
| 320 | Thomas Russell, M.B., C.M., *Glasgow*, | 1888 |
| | William Burns Macdonald, M.B., C.M., *Dunbar*, | 1888 |
| | Professor John M'Fadyean, M.B., C.M., *London*, | 1888 |
| | James W. Martin, M.D., F.R.C.P. Ed., *Dumfries*, | 1888 |
| | J. R. Home Ross, M.B., F.R.C.P. Ed., *Burmah*, | 1888 |
| 325 | John Smith, M.D., M.R.C.S. Eng., *Kirkcaldy*, | 1889 |
| | Benjamin D. C. Bell, L.R.C.P. & S. Ed., *Kirkwall*, | 1889 |
| | A. Home Douglas, M.B., F.R.C.P. Ed., *Nice*, | 1889 |
| | Surgeon-Captain C. H. Bedford, M.D., D.Sc., M.R.C.S. Eng., *Bengal Army*, | 1889 |
| | Alexander John Keiller, L.R.C.P. & S. Ed., *New Brunswick*, | 1889 |
| 330 | D. G. Braidwood, M.B., C.M., *Halkirk, Caithness*, | 1889 |
| | Professor J. Berry Haycraft, M.D., D.Sc., *Cardiff*, | 1889 |
| | Professor A. W. Hughes, M.B., F.R.C.S. Ed., M.R.C.S. Eng., *London*, | 1889 |
| | Albert E. Morison, M.B., F.R.C.S. Ed., M.R.C.S. Eng., *Hartlepool*, | 1889 |
| | William H. Barrett, M.B., C.M., *Southport*, | 1890 |
| 335 | James Hunter, M.D., C.M., *Linlithgow*, | 1890 |
| | George M. Robertson, M.B., F.R.C.P. Ed., *Larbert*, | 1890 |
| | Professor Ralph Stockman, M.D., F.R.C.P. Ed., *Glasgow*, | 1891 |
| | Charles Templeman, M.D., C.M., *Dundee*, | 1891 |
| | J. J. Douglas, M.D., F.R.C.P. Ed., *London*, | 1891 |
| 340 | Robert Stirling, M.B., C.M., *Perth*, | 1891 |
| | Simson C. Fowler, M.B., C.M., *Juniper Green*, | 1892 |
| | Robert Dundas Helm, M.D., C.M., *Carlisle*, | 1892 |
| | William Gordon Woodrow Sanders, M.B., F.R.C.P. Ed., *Caen*, | 1892 |
| | Ernest Coleman Moore, M.B., C.M., | 1892 |
| 345 | W. Ramsay Smith, M.B., C.M., *South Australia*, | 1892 |
| | T. Herbert Littlejohn, M.B., F.R.C.S. Ed., *Scarborough*, | 1892 |
| | Alexander Peyer, M.D., *Zürich*, | 1893 |
| | Alexander Reid Urquhart, M.D., F.R.C.P. Ed., *Perth*, | 1893 |
| | F. W. Foxcroft, M.D., C.M., *Wilmslow*, | 1893 |
| 350 | William B. Mackay, M.D., M.R.C.S. Eng., *Berwick-on-Tweed*, | 1893 |
| | Alex. Mitchell Stalker, M.D., C.M., *Dundee*, | 1893 |
| | D. W. Johnston, F.R.C.S. Ed., *Johannesburg, S. Africa*, | 1893 |
| | William Brendon T. Gubbin, M.D., C.M., *Bardolph, Hertford*, | 1893 |
| | Frank Ashby Elkins, M.D., C.M., *Watford*, | 1893 |
| 355 | Philip Grierson Borrowman, M.B., C.M., *Elie*, | 1893 |
| | William Craig, M.B., C.M., *Cowdenbeath*, | 1894 |
| | James Mackenzie, M.D., C.M., *Burnley*, | 1894 |
| | William Simmers, M.B., C.M., *Crail*, | 1894 |
| | Frederick Maurice Graham, F.R.C.S. Ed., L.R.C.P. & S. Ed., *Market Drayton*, | 1894 |
| 360 | Professor Robert Muir, M.D., F.R.C.P. Ed., *Glasgow*, | 1894 |
| | Gopal Govind Vatve, M.D., *Bombay*, | 1895 |
| | John Hosack Fraser, M.B., F.R.C.P. Ed., *Bridge of Allan*, | 1895 |
| | John Struthers, M.B., C.M., *South Africa*, | 1895 |
| | George Thomas Beatson, M.D., C.M., *Glasgow*, | 1895 |
| 365 | Donald Macaulay, M.B., C.M., *Johannesburg*, | 1895 |
| | Andrew Balfour, M.D., C.M., | 1895 |
| | Robert Durward Clarkson, M.B., C.M., *Falkirk*, | 1896 |

| | | Date of Admission. |
|---|---|---|
| | George Kerr Grimmer, M.B., C.M., *South Queensferry*, . | 1897 |
| | Edward Arthur Mills-Roberts, M.B., C.M., *Bangor*, . | 1897 |
| 370 | Samuel Macvie, M.B., C.M., *Chirnside*, . . . | 1897 |
| | Donald George Macleod Munro, M.B., C.M., *Cheltenham*, . | 1898 |
| | Andrew William Messer, M.B., C.M., *Scotswood-on-Tyne*, . | 1899 |
| | Samuel William Carruthers, M.D., C.M., *London*, . . | 1899 |

## ORDINARY MEMBERS.

ARRANGED ALPHABETICALLY.

### (*a.*) Members who pay the Annual Subscription of Ten Shillings.

| | | |
|---|---|---|
| | Dr R. Abernethy, 10 St Colme Street, . . . | 1892 |
| | Dr J. O. Affleck, 38 Heriot Row, . . . | 1871 |
| | Dr A. C. Ainslie, 20 Newington Road, . . . | 1898 |
| | Dr R. S. Aitchison, 74 Great King Street, . . . | 1887 |
| 5 | Dr W. A. J. Alexander, 1 George Place, Pilrig, . . | 1896 |
| | Dr John Anderson, Pitlochry, . . . . | 1896 |
| | Dr James Andrew, 2 Atholl Crescent, . . . | 1869 |
| | Professor Annandale, 34 Charlotte Square, . . . | 1863 |
| | Dr E. F. Armour, 149 Bruntsfield Place, . . . | 1890 |
| 10 | Dr G. W. Balfour, Westfield, Colinton, . . . | 1874 |
| | Dr J. W. Ballantyne, 24 Melville Street, . . . | 1885 |
| | Dr A. H. Freeland Barbour, 4 Charlotte Square, . . | 1881 |
| | Joseph Bell, Esq., 2 Melville Crescent, . . . | 1862 |
| | Dr G. A. Berry, 31 Drumsheugh Gardens, . . . | 1883 |
| 15 | Dr R. J. A. Berry, 4 Howard Place, . . . . | 1893 |
| | Dr Alexander Black, 13 Howe Street, . . . | 1883 |
| | Dr W. T. Black, 2 George Square, . . . . | 1877 |
| | Dr Robert H. Blaikie, 42 Minto Street, . . . | 1883 |
| | Dr Bleloch, 2 Lonsdale Terrace, . . . . | 1871 |
| 20 | Dr G. P. Boddie, 73 Bruntsfield Place, . . . | 1888 |
| | William Booth, Esq., 2 Minto Street, . . . | 1888 |
| | Dr F. D. Boyd, 6 Atholl Place, . . . . | 1891 |
| | Dr Byrom Bramwell, 23 Drumsheugh Gardens, . . | 1876 |
| | Dr N. T. Brewis, 23 Rutland Street, . . . . | 1886 |
| 25 | Dr J. J. Graham Brown, 3 Chester Street, *Secretary*, . . | 1878 |
| | Dr J. Murdoch Brown, 9 Walker Street, . . . | 1885 |
| | Dr Alexander Bruce, 13 Alva Street, . . . . | 1883 |
| | Dr Lewis C. Bruce, The Asylum, Murthly, Perthshire, . | 1895 |
| | Dr Buist, 1 Clifton Terrace, . . . . . | 1877 |
| 30 | Dr T. M. Burn-Murdoch, 14 Charlotte Square, . . | 1886 |
| | Dr Cadell, 22 Ainslie Place, *Vice-President*, . . . | 1870 |
| | Dr Francis M. Caird, 21 Rutland Street, . . . | 1883 |
| | Dr H. L. Calder, 60 Leith Walk, Leith, . . . | 1884 |
| | Dr James Cameron, 13 Fettes Row, . . . . | 1895 |
| 35 | Dr Edward Carmichael, 21 Abercromby Place, . . | 1887 |
| | Dr J. Carmichael, 22 Northumberland Street, . . | 1870 |
| | Dr C. W. Cathcart, 8 Randolph Crescent, . . . | 1883 |
| | Dr J. G. Cattanach, 3 Alvanley Terrace, . . . | 1895 |
| | Dr T. F. S. Caverhill, 16 Randolph Crescent, . . | 1883 |
| 40 | Professor John Chiene, 26 Charlotte Square, . . . | 1867 |
| | Dr J. A. Clark, 4 Cambridge Street, . . . . | 1893 |
| | Dr Church, 36 George Square, . . . . | 1876 |

| | | Date of Admission. |
|---|---|---|
| | Dr Clouston, Tipperlinn House, Morningside Place, | 1861 |
| | Dr Cotterill, 24 Manor Place, *Secretary*, | 1878 |
| 45 | Dr William Craig, 71 Bruntsfield Place, | 1869 |
| | Dr J. Frank Crombie, North Berwick, | 1897 |
| | Dr Halliday Croom, 25 Charlotte Square, | 1870 |
| | Dr G. Matheson Cullen, 48 Lauriston Place, | 1892 |
| | Dr A. S. Cumming, 18 Ainslie Place, | 1884 |
| 50 | Dr John Cumming, 20 Gilmore Place, | 1894 |
| | Dr R. J. B. Cunynghame, 18 Rothesay Place, | 1868 |
| | Dr T. B. Darling, 13 Merchiston Place, | 1887 |
| | Dr G. D. Darlington, 10 Ardmillan Terrace, | 1898 |
| | Dr M. Dewar, 24 Lauriston Place, | 1885 |
| 55 | Dr George Dickson, 9 India Street, | 1884 |
| | Dr C. E. Douglas, Cupar-Fife, | 1894 |
| | Dr Kenneth M. Douglas, 19 Chester Street, | 1888 |
| | Dr J. W. Dowden, 22 Melville Street, | 1893 |
| | Dr John A. H. Duncan, 32 Morningside Drive, | 1895 |
| 60 | Dr Kirk Duncanson, 22 Drumsheugh Gardens, | 1871 |
| | Dr H. M. Dunlop, 20 Abercromby Place, | 1883 |
| | Dr J. C. Dunlop, 24 Stafford Street, | 1892 |
| | Dr J. Dunsmure, 53 Queen Street, *Vice-President*, | 1872 |
| | Dr Thomas Easton, | 1894 |
| 65 | Dr George Elder, 7 Leopold Place, | 1896 |
| | Dr William Elder, 4 John's Place, Leith, | 1892 |
| | Dr R. W. Felkin, 23 Henrietta Street, Cavendish Square, London, W., | 1885 |
| | Dr J. Haig Ferguson, 25 Rutland Street, | 1885 |
| | Dr W. A. Finlay, St Helen's, Russell Place, Trinity, | 1875 |
| 70 | Dr Andrew Fleming, 8 Napier Road, | 1880 |
| | Dr R. A. Fleming, 10 Chester Street, | 1890 |
| | Dr Foulis, 34 Heriot Row, | 1875 |
| | Dr J. S. Fowler, 42 Henderson Row, | 1895 |
| | Dr John Fraser, 19 Strathearn Road, | 1878 |
| 75 | Professor Thomas R. Fraser, 13 Drumsheugh Gardens, | 1865 |
| | Dr Garland, 53 Charlotte Street, Leith, | 1873 |
| | Dr G. A. Gibson, 17 Alva Street, | 1880 |
| | Dr A. B. Giles, 1 Kew Terrace, | 1893 |
| | Dr A. Lockhart Gillespie, 23 Walker Street, | 1891 |
| 80 | Dr D. J. Graham, 8 Gilmore Place, | 1897 |
| | Dr J. Taylor Grant, 6 Coates Place, | 1898 |
| | Dr J. Allan Gray, 107 Ferry Road, | 1879 |
| | Dr T. Paul Gray, 6 St Catherine's Place, | 1899 |
| | Professor Greenfield, 7 Heriot Row, | 1886 |
| 85 | Dr David M. Greig, 25 Tay Street, Dundee, | 1892 |
| | Dr G. L. Gulland, 6 Alva Street, | 1888 |
| | Dr A. C. Guthrie, 171 Constitution Street, Leith, | 1890 |
| | Dr William Guy, 11 Wemyss Place, | 1890 |
| | Dr William Haldane, Viewforth, Bridge of Allan, | 1889 |
| 90 | Dr John Hardie, Stogursey, near Bridgewater, | 1895 |
| | Dr D. Berry Hart, 29 Charlotte Square, | 1886 |
| | Dr James Harvey, 7 Blenheim Place, | 1893 |
| | Dr Henry Hay, 7 Brandon Street, | 1884 |
| | Dr John Henderson, 7 John's Place, Leith, | 1848 |
| 95 | Mr J. W. B. Hodsdon, 6 Chester Street, | 1883 |
| | Dr George Hunter, 33 Palmerston Place, | 1876 |
| | Dr Husband, 4 Royal Circus, | 1849 |
| | Dr J. Hutcheson, 44 Moray Place, | 1890 |
| | Dr Robert Inch, Gorebridge, | 1887 |
| 100 | Dr R. W. Inkster, 31 Windsor Street, | 1895 |
| | Dr W. Wotherspoon Ireland, Mavisbush House, Polton, | 1893 |
| | Dr Alex. James, 10 Melville Crescent, | 1877 |
| | Dr Allan Jamieson, 35 Charlotte Square, | 1876 |

| | | Date of Admission. |
|---|---|---|
| | Dr Hugh Jamieson, 13 Rutland Street, | 1889 |
| 105 | Dr James Jamieson, 43 George Square, | 1877 |
| | Dr R. J. Johnston, 1 Buccleuch Place, | 1897 |
| | Dr G. M. Johnston, 7 Wellington Place, Leith, | 1888 |
| | Dr R. M'Kenzie Johnston, 2 Drumsheugh Gardens, *Treasurer*, | 1883 |
| | Dr C. Kennedy, 5 Salisbury Road, | 1886 |
| 110 | Dr C. B. Ker, City Fever Hospital, | 1894 |
| | Dr George Kerr, 6 St Colme Street, | 1885 |
| | Dr J. H. A. Laing, 11 Melville Street, | 1889 |
| | Dr Linton, 60 George Square, | 1863 |
| | Dr Harvey Littlejohn, 11 Stafford Street, | 1889 |
| 115 | Professor Sir Henry D. Littlejohn, 24 Royal Circus, | 1853 |
| | Dr R. A. Lundie, 55A Grange Road, | 1883 |
| | Dr P. M'Bride, 16 Chester Street, | 1879 |
| | Dr W. A. M'Cutchan, County and City Asylum, Burghill, Hereford, | 1897 |
| | Dr William C. M'Ewan, Prestonpans, | 1892 |
| 120 | John M'Gibbon, Esq., 55 Queen Street, | 1868 |
| | Dr MacGillivray, 15 Charlotte Square, | 1877 |
| | Dr F. Reid Mackay, 5 Walker Street, | 1899 |
| | Dr G. Mackay, 74 Bruntsfield Place, | 1878 |
| | Dr George Mackay, 20 Drumsheugh Gardens, | 1887 |
| 125 | Dr Robert Mackenzie, Napier Villa, Merchiston, | 1893 |
| | Dr W. Leslie Mackenzie, 4 Summerside Place, Leith, | 1895 |
| | Dr William A. Mackintosh, 13 Abercromby Place, Stirling, | 1896 |
| | Professor Sir Douglas Maclagan, 28 Heriot Row, | 1834 |
| | Dr J. Shaw M'Laren, 14 Walker Street, | 1887 |
| 130 | Dr P. Hume Maclaren, 1 Drumsheugh Gardens, | 1868 |
| | Dr John Macmillan, 27 Warrender Park Road, | 1897 |
| | Dr John Macpherson, 8 Darnaway Street, | 1891 |
| | Dr John MacRae, Lynwood, Murrayfield, | 1893 |
| | Dr D. Menzies, 20 Rutland Square, | 1878 |
| 135 | Dr James Middlemass, Royal Asylum, Morningside, | 1895 |
| | Dr A. Miles, 1 George Square, | 1892 |
| | Dr J. Mill, 26 N. Fort Street, Leith, | 1886 |
| | A. G. Miller, Esq., 7 Coates Crescent, | 1867 |
| | Dr W. H. Miller, 51 Northumberland Street, | 1895 |
| 140 | Dr D. Milligan, 11 Palmerston Place, | 1885 |
| | Sir Arthur Mitchell, 34 Drummond Place, | 1859 |
| | Dr James Mowat, 1 Priestfield Road, | 1893 |
| | Dr John Mowat, 6 Buccleuch Place, | 1885 |
| | Dr Claud Muirhead, 30 Charlotte Square, | 1866 |
| 145 | Dr R. Milne Murray, 11 Chester Street, | 1884 |
| | Dr P. Orphoot, 113 George Street, | 1865 |
| | Dr John Orr, 1 Rillbank Crescent, | 1895 |
| | Dr W. Basil Orr, 13 Braid Road, | 1893 |
| | Dr G. Keppie Paterson, 19 Albany Street, | 1889 |
| 150 | Dr D. Noel Paton, 22 Lynedoch Place, | 1885 |
| | Dr H. A. Peddie, 24 Palmerston Place, | 1893 |
| | Dr R. W. Philip, 45 Charlotte Square, | 1883 |
| | Dr Playfair, 5 Melville Crescent, | 1874 |
| | Dr T. Proudfoot, 30 Lauriston Place, | 1889 |
| 155 | Dr R. B. Purves, 17 Walker Street, | 1899 |
| | Dr Harry Rainy, 25 George Square, | 1866 |
| | Dr Rattray, Portobello, | 1874 |
| | Dr James Ritchie, 22 Charlotte Square, | 1873 |
| | Dr R. Peel Ritchie, 1 Melville Crescent, | 1862 |
| 160 | Dr Argyll Robertson, 18 Charlotte Square, | 1861 |
| | Dr William F. Robertson, Charterhall Road, | 1893 |
| | Dr W. G. Aitchison Robertson, 26 Minto Street, | 1892 |
| | Dr T. R. Ronaldson, 8 Charlotte Square, | 1877 |
| | Dr R. M. Ronaldson, 17 Morningside Road, | 1895 |

| | | Date of Admission. |
|---|---|---|
| 165 | Dr William Russell, 3 Walker Street, | 1884 |
| | Dr E. G. Salt, 50 George Square, | 1895 |
| | Dr James Scott, 43 Minto Street, | 1895 |
| | Dr Andrew Semple, 10 Forres Street, | 1883 |
| | Dr Theodore Shennan, 71 Leamington Terrace, | 1897 |
| 170 | Sir John Sibbald, 18 Great King Street, | 1859 |
| | Professor Simpson, 52 Queen Street, | 1859 |
| | Dr A. A. Scot Skirving, 29 Drummond Place, | 1897 |
| | Dr A. T. Sloan, 22 Forth Street, | 1885 |
| | Dr Andrew Smart, 15 Rutland Square, | 1865 |
| 175 | Dr G. D. Smith, 148 Ferry Road, | 1877 |
| | Dr James Smith, 1 Parson's Green Terrace, | 1891 |
| | Dr John Smith, 11 Wemyss Place, | 1856 |
| | Dr J. Cormack Smith, 9 Brunton Place, | 1896 |
| | Dr William Smith, 14 Hartington Gardens, | 1890 |
| 180 | Dr John Stevens, 2 Shandon Street, | 1892 |
| | Professor Sir T. Grainger Stewart, 19 Charlotte Square, | 1861 |
| | Dr J. S. Stewart, 15 Merchiston Place, | 1885 |
| | Dr Robert Stewart, 42 George Square, | 1892 |
| | Dr William Stewart, 146 Ferry Road, Leith, | 1889 |
| 185 | Dr H. J. Stiles, 8 St Colme Street, | 1889 |
| | Dr S. Stirling, 4 Coates Crescent, | 1891 |
| | Dr A. Stodart-Walker, 30 Walker Street, | 1894 |
| | Dr John Strachan, Dollar, | 1867 |
| | Dr C. A. Sturrock, Dunfermline, | 1894 |
| 190 | Dr Allan C. Sym, 144 Morningside Road, | 1889 |
| | Dr William G. Sym, 20 Alva Street, | 1889 |
| | Dr W. Taylor, 12 Melville Street, | 1871 |
| | Dr C. C. Teacher, 8 Nile Grove, | 1887 |
| | Dr C. H. Thatcher, 8 Melville Crescent, | 1876 |
| 195 | Dr R. Thin, 38 Albany Street, | 1890 |
| | Dr Alexis Thomson, 32 Rutland Square, | 1887 |
| | Dr John Thomson, 14 Coates Crescent, | 1887 |
| | Dr T. J. Thyne, 2 Dean Terrace, | 1898 |
| | Dr John Tod, 93 Ferry Road, Leith, | 1895 |
| 200 | Sir John Batty Tuke, 20 Charlotte Square, *President*, | 1864 |
| | Dr F. J. Turnbull, 6 Randolph Place, | 1896 |
| | Dr Dawson Turner, 37 George Square, | 1890 |
| | Dr Logan Turner, 20 Coates Crescent, | 1892 |
| | Dr Underhill, 8 Coates Crescent, | 1872 |
| 205 | Dr Norman Walker, 7 Manor Place, | 1891 |
| | Dr D. Wallace, 11 Rutland Street, | 1887 |
| | Dr Douglas C. Watson, 22 Coates Crescent | 1894 |
| | Dr P. H. Watson, 16 Charlotte Square, | 1856 |
| | Dr N. P. Watt, 1 Denham Green Terrace, Trinity, | 1898 |
| 210 | Dr A. D. Webster, Belleville Lodge, Blacket Avenue, | 1883 |
| | Dr George R. Wilson, Mavisbank, Polton, | 1892 |
| | J. L. Wilson, Esq., 4 Buccleuch Place, | 1883 |
| | Dr T. D. Wilson, 10 Newington Road, | 1880 |
| | Dr Russell E. Wood, 9 Darnaway Street, | 1883 |
| 215 | Dr W. Fraser Wright, Bonnington Mount, Bonnington Ter., | 1894 |
| | Dr John Wyllie, 44 Charlotte Square, | 1868 |
| | Dr P. A. Young, 25 Manor Place, | 1870 |
| | Dr R. J. Erskine Young, 8 Alva Street, | 1892 |

## (*b.*) Members Exempted under Rule V. from paying the Annual Subscription.

| | | Date of Admission. |
|---|---|---|
| | Dr D. Aitken, 152 *Packington Street, Islington, London, N.*, | 1887 |
| 220 | Dr D. H. Anderson, 131 *Abbey Road, Barrow-in-Furness*, | 1887 |
| | Dr Archibald, 2 *The Avenue, Beckenham, Kent*, | 1882 |
| | Dr J. A. Armitage, 28 *Waterloo Road South, Wolverhampton*, | 1887 |
| | Dr W. Badger, *Penicuik*, | 1882 |
| | Dr Edwin Baily, *Oban*, | 1883 |
| 225 | Dr Andrew Balfour, *Portobello*, | 1874 |
| | Dr Andrew Balfour, 21 *Heriot Row*, | 1895 |
| | Dr James Craig Balfour, *Westfield, Colinton*, | 1884 |
| | Dr J. H. Balfour, *Portobello*, | 1881 |
| | Dr Alexander Ballantyne, *Dalkeith*, | 1872 |
| 230 | Dr W. H. Barrett, 29 *Park Crescent, Hesketh Park, Southport*, | 1890 |
| | Dr George T. Beatson, 7 *Woodside Crescent, Glasgow*, | 1895 |
| | Surgeon-Captain C. H. Bedford, *Bengal Army, care of W. Watson & Co.*, 28 *Apollo Street, Bombay*, | 1889 |
| | Dr Benjamin D. C. Bell, *Kirkwall*, | 1889 |
| | Dr G. J. H. Bell, *Bengal Army*, | 1884 |
| 235 | Dr G. H. Bentley, *Kirkliston*, | 1877 |
| | Dr J. S. Beveridge, *Foulsham, Norfolk*, | 1861 |
| | Dr J. W. Black, 15 *Clarges Street, Piccadilly, London, W.*, | 1865 |
| | Dr P. G. Borrowman, *Elie, Fife*, | 1893 |
| | Dr D. G. Braidwood, *Halkirk, Caithness*, | 1889 |
| 240 | Dr David Brodie, *Ventnor House, Canterbury*, | 1865 |
| | Dr Andrew Brown, 1 *Bartholomew Road, Kentish Town, London, N.W.*, | 1884 |
| | Dr John Brown. | 1878 |
| | Dr J. Macdonald Brown, 5 *Lymington Road, West Hampstead, London, N.W.*, | 1883 |
| | Dr S. W. Carruthers, 44 *Central Hill, Norwood, London, S.E.*, | 1899 |
| 245 | Dr R. D. Clarkson, *Falkirk*, | 1896 |
| | Professor Cleland, *The University, Glasgow*, | 1864 |
| | Dr A. R. Coldstream, 24 *Lung Arno Nuovo, Florence, Italy*, | 1878 |
| | Dr John Connel, *Peebles*, | 1876 |
| | Dr William Craig, *Cowdenbeath, Fife*, | 1894 |
| 250 | Dr J. R. Crease, 2 *Ogle Terrace, South Shields*, | 1885 |
| | Dr P. M. Deas, *Wonford House, Exeter*, | 1868 |
| | Dr Archibald Dickson, *Hartree House, Biggar*, | 1871 |
| | Dr A. Halliday Douglas, *The Neuk, Peebles*, | 1842 |
| | Dr A. Home Douglas, 5 *Rue de Russie, Nice, France*, | 1889 |
| 255 | Dr J. J. Douglas, 42 *Centrall Hill, Upper Norwood, London*, | 1891 |
| * | Dr W. B. Dow, *Dunfermline*, | 1879 |
| | Dr J. W. Eastwood, *Dinsdale Park, Darlington*, | 1871 |
| | Dr F. A. Elkins, *The Asylum, Leavesden, near King's Langley*, | 1893 |
| | Sir R. B. Finlay, Q.C., M.P., *Middle Temple, London*, | 1864 |
| 260 | Dr Simson C. Fowler, *Juniper Green*, | 1892 |
| | Dr F. W. Foxcroft, 33 *Paradise Street, Birmingham*, | 1893 |
| | Dr Dyce Fraser, 1 *Oxford Villas, Teddington*, | 1883 |
| | Dr J. Hosack Fraser, *Bellfield, Bridge of Allan*, | 1895 |
| | Professor Gamgee, 8 *Avenue de la Garve, Lausanne, Switzerland*, | 1863 |
| 265 | Dr William Gayton, *Bartram Lodge, Fleet Road, Hampstead, London, N.W.*, | 1886 |
| | Dr F. M. Graham, *Willowbridge, Market Drayton*, | 1894 |
| | Dr W. C. Greig, *Otago, New Zealand*, | 1884 |

| | | Date of Admission. |
|---|---|---|
| | Dr George K. Grimmer, *South Queensferry*, | 1897 |
| | Dr Groesbeck, *Cincinnati*, | 1875 |
| 270 | Dr W. B. T. Gubbin, *Redland, Bristol*, | 1893 |
| | His Excellency Dr R. H. Gunning, 12 *Addison Crescent, Kensington, London, W.*, | 1846 |
| | Dr John Haddon, *Denholm, Roxburghshire*, | 1883 |
| | Dr Archibald Hall, *Montreal*, | 1853 |
| | Professor D. J. Hamilton, *The University, Aberdeen*, | 1876 |
| 275 | Dr J. W. Hamp, *Penn Road, Wolverhampton*, | 1887 |
| | Dr A. W. Hare, *Roche, Cornwall*, | 1883 |
| | Dr J. Home-Hay, *Alloa*, | 1880 |
| | Professor J. Berry Haycraft, 1 *St Andrew's Place, Cardiff*, | 1889 |
| | Dr Stanley Haynes, *Malvern, Worcestershire*, | 1864 |
| 280 | Dr R. Dundas Helm, 3 *Alfred Street N., Portland Square, Carlisle*, | 1892 |
| | Dr R. E. Horsley, *Jud-Falls, Stoneyhurst, Lancashire*, | 1886 |
| | Professor A. W. Hughes, *King's College, London*, | 1889 |
| | Dr James Hunter, *St Catherine's, Linlithgow*, | 1890 |
| | Dr W. Hunter, 103 *Harley Street, Cavendish Square, London, W.*, | 1887 |
| 285 | Dr J. Carlyle Johnstone, *The Asylum, Melrose*, | 1882 |
| | Dr D. W. Johnston, *P.O. Box* 2022, *Johannesburg, South Africa*, | 1893 |
| | Dr James Johnston, 53 *Princes Square, Bayswater, London, W.*, | 1871 |
| | Dr J. Keay, *District Asylum, Inverness*, | 1887 |
| | Dr A. J. Keiller, *New Brunswick, Canada*, | 1889 |
| 290 | Dr George Keith, *Moidart Cottage, Currie*, | 1845 |
| | Dr Skene Keith, 42 *Charles Street, Berkeley Square, London, W.*, | 1885 |
| | Dr W. Scott Lang, | 1886 |
| | Professor R. F. C. Leith, M.B., *Mason University College, Birmingham*, | 1886 |
| | Dr Herbert Littlejohn, *Scarborough*, | 1892 |
| 295 | Dr W. H. Lowe, *Woodcote Lodge, Inner Park, Wimbledon, Surrey*, | 1845 |
| | Dr Robert Lucas, *Dalkeith*, | 1875 |
| | Dr Donald Macaulay, *Jumpers' Gold Mining Co., P.O. Box* 1043, *Johannesburg*, | 1895 |
| | Dr F. R. Macdonald, *Inveraray*, | 1860 |
| | Dr K. N. Macdonald, 21 *Clarendon Crescent*, | 1880 |
| 300 | Dr W. B. Macdonald, *Dunbar*, | 1888 |
| | Dr John A. Macdougall, *Cannes, France*, | 1875 |
| | Professor J. M'Fadyean, 101 *Great Russell Street, London, W.C.*, | 1888 |
| | Dr John Mackay, *Aberfeldy*, | 1881 |
| | Dr W. B. Mackay, 23 *Castlegate, Berwick-on-Tweed*, | 1893 |
| 305 | Professor M'Kendrick, *The University, Glasgow*, | 1870 |
| | Dr James Mackenzie, 66 *Bank Parade, Burnley, Lancashire*, | 1894 |
| | Dr T. J. Maclagan, 9 *Cadogan Place, Belgrave Square, London, S.W.*, | 1875 |
| | Dr Roderick M'Laren, 23 *Portland Square, Carlisle*, | 1882 |
| | Dr S. Macvie, *Chirnside*, | 1897 |
| 310 | Dr J. W. Martin, *Burnfoot, Cluden, Dumfries*, | 1888 |
| | Dr A. Matthew, *Corstorphine*, | 1882 |
| | Dr Andrew W. Messer, *Lenington, Scotswood-on-Tyne*, | 1899 |
| | Dr E. A. Mills-Roberts, *Bryn Meurig, Bethesda, Bangor*, | 1897 |
| | Dr J. Moolman, *Cape of Good Hope*, | 1877 |
| 315 | Dr E. C. Moore, 2 *Coates Place*, | 1892 |
| | Dr A. E. Morison, *Brougham Terrace, Hartlepool*, | 1889 |
| | Dr J. Rutherford Morison, 14 *Saville Row, Newcastle-on-Tyne*, | 1882 |

| | | Date of Admission. |
|---|---|---|
| | Professor Robert Muir, *The University, Glasgow*, | 1894 |
| | Dr D. G. Macleod Munro, 1 *Oriel Villas, Cheltenham*, | 1898 |
| 320 | Dr J. Ivor Murray, 2 *Pavilion Square, Scarboro'*, | 1857 |
| | Dr Andrew Scott Myrtle, *Harrogate*, | 1859 |
| | Dr Leith Napier, *The General Hospital, Adelaide, South Australia*, | 1879 |
| | Dr T. Goodall Nasmyth, *Cupar-Fife*, | 1884 |
| | Dr Ernest F. Neve, *Srinagar, Kashmir, India*, | 1884 |
| 325 | Dr T. Wyld Pairman, *H.M. Prison, Lyttelton, New Zealand*, | 1884 |
| | Professor Bell Pettigrew, *St Andrews*, | 1873 |
| | Dr Alexander Peyer, *Zürich*, | 1893 |
| | Dr J. A. Philip, *Rue Victor Hugo, Boulogne-Sur-Mer, France*, | 1878 |
| | Professor W. S. Playfair, 38 *Grosvenor Street, London, W.*, | 1857 |
| 330 | Sir W. O. Priestley, M.P., 17 *Hertford Street, Mayfair, London, W.*, | 1854 |
| | Dr J. H. Pringle, 172 *Bath Street, Glasgow*, | 1886 |
| | Dr S. Hale Puckle, *Bishop's Castle*, | 1885 |
| | Dr G. M. Robertson, *The Copse, Larbert*, | 1890 |
| | Dr J. Maxwell Ross, *Avenel, Maxwelltown, Dumfries*. | 1882 |
| 335 | Dr J. R. Home Ross, *Burmah*, | 1888 |
| | Dr Joseph C. Ross, *Withington*, | 1884 |
| | Dr S. Rumboll, *Hope Villa, Hillary Place, Leeds*, | 1887 |
| | Dr Thomas Russell, 27A *Westmuir Street, Parkhead, Glasgow*, | 1888 |
| | Dr Gordon Sanders, *Cannes, France*, | 1892 |
| 340 | Dr F. A. Saunders, *Grahamstown, South Africa*, | 1884 |
| | Dr Thomas R. Scott, *Musselburgh*, | 1884 |
| | Dr C. A. E. Sheaf, *Ovalu, Fiji*, | 1871 |
| | Dr G. F. Shiels, 229 *George Street, San Francisco*, | 1887 |
| | Dr W. H. Shirreff, *Port Fairy, Victoria, Australia*, | 1883 |
| 345 | Dr William Simmers, *Denburn, Crail*, | 1894 |
| | Dr T. Skinner, 6 *York Place, Portman Square, London, W.*, | 1856 |
| | Dr John Smith, *Brycehall, Kirkcaldy*, | 1889 |
| | Dr W. Ramsay Smith, *The General Hospital, Adelaide, South Australia*, | 1892 |
| | Dr Van Someren, *Goldhurst Terrace, South Hampstead, London, N.W.*, | 1845 |
| 350 | Dr Somerville, *Galashiels*, | 1877 |
| | Dr A. M. Stalker, 140 *Nethergate, Dundee*, | 1893 |
| | Dr Graham Steell, 96 *Moseley Street, Manchester*, | 1877 |
| | Professor Stephenson, *University, Aberdeen*, | 1861 |
| | Dr Robert Stirling, 4 *Atholl Place, Perth*, | 1891 |
| 355 | Professor Ralph Stockman, *The University, Glasgow*, | 1891 |
| | Dr H. R. Storer, *Newport, Rhode Island, U.S.*, | 1855 |
| | Dr John Struthers, *Ngamakwe, Transkei, South Africa*, | 1895 |
| | Dr J. F. Sturrock, *Arima, Broughty Ferry*, | 1887 |
| | Professor J. Symington, *Queen's College, Belfast*, | 1878 |
| 360 | Dr C. Templeman, *Sanitary Office, Bell Street, Dundee*, | 1891 |
| | Dr Thin, 63 *Harley Street, Cavendish Square, London, W.*, | 1861 |
| | Dr Alexander Thom, *Viewfield, Crieff*, | 1884 |
| | Dr J. Stitt Thomson, *The Mount, Lincoln*, | 1877 |
| | Dr J. Batty Tuke, jr., *Balgreen, Murrayfield*, | 1886 |
| 365 | Dr R. S. Turner, *Keith*. | 1867 |
| | Dr T. Edgar Underhill, *Dunedin, Burnt Green, Worcestershire*, | 1885 |
| | Dr A. R. Urquhart, *Murray House, Perth*, | 1893 |
| | Dr Gopal Govind Vatve, *care of H.H. the Rajah of Miraj, Bombay, India*, | 1895 |
| | Dr A. B. Whitton, *Aberchirder*, | 1886 |
| 370 | Dr Oswald G. Wood, *Indian Army*, | 1886 |

| | Date of Admission. |
|---|---|
| Professor G. Sims Woodhead, *The University, Cambridge*, . | 1883 |
| Dr Yellowlees, *Gartnavel Asylum, Glasgow*, . . . | 1862 |
| Professor John Young, *The University, Glasgow*, . . | 1860 |

N.B.—*Members are requested to communicate with the Secretaries if they discover any errors or omissions in the List, and also to intimate all changes in their addresses.*

# CONTENTS.

## I.—ORIGINAL COMMUNICATIONS.

### (*a.*) Histological.



### (*b.*) Toxicological.



### (*c.*) Medical.

#### (1.) *General Diseases.*



#### (2.) *Fevers.*



#### (3.) *Diseases of the Nervous System.*

# TRANSACTIONS

OF THE

# MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH

FOR SESSION LXXVIII., 1898-99

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## Meeting I.—November 2nd, 1898

Dr William Craig *in the Chair*

### I. Election of Office-Bearers

The following gentlemen were elected office-bearers:—*President*, Sir John Batty Tuke; *Vice-Presidents*, Dr Francis Cadell, Dr P. A. Young, and Dr James Dunsmure; *Council*, Drs Argyll Robertson, Michael Dewar, John Thomson, J. Shaw M'Laren, H. Melville Dunlop, George Mackay, H. Alexis Thomson, and George L. Gulland; *Treasurer*, Dr M'Kenzie Johnston; *Secretaries*, Dr Graham Brown and Mr J. M. Cotterill; *Editor of Transactions*, Dr William Craig.

### II. Exhibition of Patients

1. *Mr Alexis Thomson* exhibited a patient in whom he had removed a considerable portion of the pyloric end of the stomach for cancer in July 1898. The patient, a woman aged 49, had suffered from gastric symptoms for eight months and had presented a visible and palpable tumour in the epigastrium for two months. The diagnosis of pyloric cancer had been made by Dr John Steven and confirmed by Dr

Murdoch Brown. The operation was performed by Kocher's method; the Murphy button, with which the junction between the stomach and duodenum was completed, was passed on the 13th day. The patient had gained 2 stones 4 lbs. in weight, was now the picture of health, and was able to indulge freely in ordinary diet.

2. *Dr Allan Jamieson* exhibited a case of TYLOSIS AND NÆVUS NEUROTICUS. Nævus neuroticus, or an acanthosis of congenital origin, though often progressive during later life, and frequently following one or more nerves in their cutaneous distribution, on one or both sides, is regarded as a manifestation of ichthyosis, by many authors. The condition known as Tylosis, a horny overgrowth of the epidermis of the palms and soles, is not so widely admitted as a variety of ichthyosis, though so regarded by him (Dr Allan Jamieson). The case he now has the honour of showing exhibits both anomalies, and thus may be held to support his view. The skin of the greater part of both palms of this young woman, aged 18, though looking older, was enormously thickened on her admission to Ward 38, on the 29th September 1898. The surface showed the yellowish tint so characteristic of epidermic overgrowth in this region, while the palms perspired profusely. The chief amount of thickening occurred along the palmar surface of the fingers, and on the side and ball of the thumbs and ball of the little finger. There was less hypertrophy on the left than on the right. The condition almost entirely incapacitated her for work as a domestic servant. The greater part of the dorsal surface of the hands and the forearms all round presented an appearance like soft plush or fine short cut velvet. This corresponded to the modification of structure found in nævus neuroticus, though it is more usual to have an arrangement in lines or dots, than over a continuous area, as here. The treatment has consisted in the application of a ten per cent. resorcin plaster muslin to the palms, the soddened epidermis being rubbed down with pumice stone or scraped with the sharp spoon each time the plaster is changed, commonly every second day. In this way a very marked improvement has taken place, but it remains to be seen whether this will be permanent when treatment is intermitted. Nothing so far has been done for the back of the

hands and arms as these do not constitute anything more than a disfigurement.

## III. Exhibition of Specimens

*Dr Aitchison Robertson* exhibited a large TONSILLAR CALCULUS, and stated that the occurrence of large calculi in the tonsils is a condition so relatively rare that it merits a description of the following case :—

The patient is a healthy man, aged 50. About six years ago he began to suffer from repeated attacks of tonsillitis, which often ended in suppuration. His occupation as reader in a large newspaper office exposed him to chills. These inflammatory attacks laid him up repeatedly, and especially during spring and autumn. About three years ago the submaxillary glands of his right side began to enlarge and become painful. They ultimately suppurated and discharged by a small sinus in front of the neck at the level of the thyroid cartilage. This discharge continued more or less persistently for several months until the outflow was obstructed in some way, when a diffuse cellulitis of the neck set in. This extended from the right ear forwards and reached as far downwards as the middle of the sternum ; his temperature rose and the breathing became somewhat embarrassed. The sinus was therefore slit up as far as the glands and a large quantity of pus evacuated. The patient made a rapid recovery from the operation, and since then has had immunity from further attacks of tonsillitis. For the last two years his health has been remarkably good.

At the end of June of the present year while spending his holiday at the seaside, he thought that he had caught a slight cold as he experienced a fulness in the throat. He noticed that he could not drink fluids so rapidly as before and that he had to take them in sips. On the third of July he retired to rest feeling perfectly well. During the night he was awakened, however, by a feeling of suffocation. He made a strong inspiratory effort and was nearly choked by some large body in his throat. He sprang out of bed thinking that his false teeth had slipped back, and coughing violently a large elongated somewhat oval stone was ejected. There was no bleeding either previously or at the time of expulsion of the stone. On palpating the throat a few days subsequently a rough tuberculated

area was felt behind the right pillar of the fauces and in the centre of this a depression from which the calculus had evidently been extruded. A gland on the right side below the angle of the jaw was enlarged and hard.

The calculus is somewhat oval in shape and corresponds in size to a rather small hen's egg. One extremity is a good deal broader than the other. It is of a pale yellow colour, and though the surface is fairly smooth it presents a worm-eaten appearance. When fresh, it had a very disagreeable smell, reminding one of rotten cheese. Its greatest length measures $1\frac{3}{4}$ inches and greatest breadth $1\frac{1}{2}$ inches (corresponding to 42 and 36 millimetres respectively), and weighs almost one ounce (26·8 grammes). It is arranged in concentric layers.

This is by far the largest tonsillar calculus which he (Dr Aitchison Robertson) has been able to find any record of. The most of those described have varied from the size of a pea to that of a small nut. Dr Nixon (1) removed a calculus as large as the first joint of the thumb from the tonsil of a young man, but this standard of comparison is unfortunately very variable.

As regards their *ætiology*, such calculi seem to occur in persons who have suffered from repeated attacks of tonsillitis. In most of the cases there is a history of suppuration of the tonsil either precedent to or co-existent with the extrusion of the calculus. When once the concretion has formed, however, it may itself be the cause of the repeated tonsillitis. In three cases related by Alexander Munro the attacks of tonsillitis were extremely frequent. One of these cases was that of an old woman who had suffered constantly for twenty years from inflammation and suppuration of the tonsil with quiescent intervals seldom exceeding three months.

In other cases, however, the tonsillar abscess has discharged itself long before the expulsion of the stone, as happened in this case. Dr Hewitt (2) narrates the case of a woman who nine years previously had suffered from suppuration of her left tonsil, and from which a calculus the size of a field bean had been expelled. Six years subsequent to this attack, and during pregnancy, two smaller calculi were expelled without previous inflammation or suppuration, and two years later during a second pregnancy the glands of her neck swelled greatly, and one month after delivery the left tonsil again suppurated and a stone weighing eleven grains was extruded.

The concretion in such cases remains evidently for long without giving rise to any disagreeable symptoms. When, however, such a tonsil becomes inflamed as a result of cold or other cause, the symptoms may rapidly become severe with marked dyspnœa and dysphagia. Dr Baker (3) records the case of a highly scrofulous woman, aged 43, who was attacked with severe sore throat, accompanied by great pain in the ear. The throat rapidly swelled, and she experienced great pain and difficulty in swallowing, accompanied by a choking sensation. During violent coughing the tonsillar abscess burst, and a calculus as large as a pea was discharged from the right tonsil.

In Dr Bryant's (4) case the patient had suffered from sore throat eighteen months previously. All the symptoms had, however, passed off, and little inconvenience was felt. The right tonsil was large, firm and painless. While coughing the patient felt sudden pain in the right side of the throat and at the same time a hard and rough calculus the size of a small nut came away. The patient of Dr Nixon's was a young man, aged 25. He had sung in public two days previous to the expulsion of the calculus and had felt no inconvenience from the large size of the concretion in the left tonsil until attacked by the general inflammation of the throat due to cold.

*Origin of these calculi.*—Some authors affirm that they are of gouty origin, but in most of the cases related no tendency to this affection was present. The father of this patient did certainly suffer from gouty troubles, but the son has up till the present at least shown no signs of this affection. Other writers think that they are formed by a deposit from the buccal secretion, and that they are similar in nature to the tartar which accumulates round the bases of the teeth. Robin (5) suggests that they may originate in small calcareous crystals, which may be seen, he says, in the centre of the tonsil.

In all probability, however, the smaller concretions are due to the retention of caseous material in the enlarged follicles of the tonsils. When not extruded, this caseous matter dries up and forms a nucleus, round which inorganic salts are deposited or the cheesy matter may be itself replaced by lime salts. It is highly improbable that they can have their origin in the retention of particles of food in the cup-shaped depressions left in the tonsil after an attack of follicular tonsillitis. It has been suggested that these particles of food undergo subsequent calcification.

The larger calculi probably result from the retention of pus in the interior of the tonsil. The abscess has either never broken or has imperfectly discharged itself and the residual matter has undergone caseation and subsequent calcification. Such stones when retained may grow in size by a further gradual deposit of lime salts until it may attain the enormous size of the stone from this case.

*Size, shape, and colour of concretions.*—The calculi are generally small in size and are compared to grains of barley. The larger ones on account of their rarity have usually been reported in the medical journals. Calculi thus noted have been said to be equal in size to a pea, bean, small nut, olive kernel, and first joint of thumb. The weight when given has varied from $3\frac{1}{2}$ to 11 grains or from 0·04 to 3 grammes.

Their shape is generally elongated. Some are described as pyriform. They are usually more or less rough on the surface, and in some cases this is so marked that they are said to resemble small mulberry calculi. One which is described by Dr Yearsley resembled a piece of rock coral in the irregularity of its shape. The *odour* which they give off when fresh is disagreeable and fœtid. When they are dry, however, it resembles that of old bone.

In *colour* they are pale yellow or light brown.

*Chemical composition.*—Their chemical composition is rarely given. Baker states that the concretion which he had examined was composed chiefly of carbonate of lime, with some oxalate of lime and animal matter. Bryant says that carbonate and phosphate of lime are the chief salts present. The only accurate analysis of a tonsillar concretion is that given by Robin as a result of Langier's analysis :—

| | |
|---|---|
| Water, . . . | 25 per cent. |
| Phosphate of lime, . | 50 „ |
| Carbonate of lime, . | 12·5 „ |
| Mucus, . . | 12·5 „ |
| | 100 per cent. |

His analysis of the calculus in this case, and in which he had the invaluable help of Dr Milroy of the Physiological Laboratory, showed that the greater part of the stone was

composed of phosphate of lime and magnesia along with a small amount of carbonate. The figures are as follows :—

| | |
|---|---|
| Organic matter, . . | 18·4 per cent. |
| Inorganic matter, . . | 81·6 „ |
| Phosphoric anhydride, . . | 50·0 „ |
| Calcium and magnesium oxides, | 28·20 „ |

*Tonsil affected.*—There does not seem to be any special tendency for one tonsil to be affected more than the other. In fourteen recorded cases, right and left tonsil were in equal proportion the seat of the calculus. In several of these cases both tonsils were enlarged at the same time.

*Hereditary predisposition.*—It is hardly possible to say anything definite regarding the influence of heredity as a predisposing cause, as the recorded cases are so few, and information on this point is seldom given in those which are reported. In the three cases of tonsillar calculus mentioned by Munro, the disease affected two sisters and a near relative. A daughter of this patient suffers from chronic sore throat with enlargement of the tonsils.

*Diagnosis.*—Unless in very evident cases where the concretion is near the surface, diagnosis must always be difficult. Patients, however, who suffer from repeated attacks of angina, with only short intervals of comparative ease, and whose tonsils are persistently of large size, form fit subjects for the development of tonsillar concretions. In such cases we should make a thorough examination of the throat by palpation. If there be any yellowish ulcers on the surface of such chronically inflamed tonsils, the probe should be used. If a calculus be present it will thus be readily detected.

*Treatment.*—As soon as the exact condition is recognised, the concretion ought to be removed, as dangerous symptoms may suddenly supervene. If the stone be large and be spontaneously evacuated, there is the possible danger of suffocation, as nearly happened with this patient. Had the stone been only a little smaller, it might have become firmly impacted in the man's throat as a result of his sudden inspiratory effort, and asphyxia might have resulted.

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(4) Bryant, *Lancet*, No. 17, 1860, ii. p. 487.
(5) Robin, "Traité des Humeurs," 2nd edit., 1874, p. 551.
(6) Terrillon, "Des Accidents causés par les calculs de l'Amygdule," *Archiv. Générales de Médicine*, Aug. 1886, ii. p. 129.
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## IV. Original Communications

## 1. TUBERCULAR NEURITIS

By Alexander James, M.D., F.R.C.P.Ed., Physician to the Royal Infirmary, and Lecturer on Practice of Medicine, Royal Colleges School of Medicine, Edinburgh

In this paper I would premise, in the first place, that by tubercular neuritis I mean a disturbance in the nutritive condition not of the peripheral nerves only, but of any part of the nervous tissue, as the result of the circulation through it of a toxin developed by tubercular disease. Such conditions are not very common, yet they are not at all infrequent, and as their recognition is as important as their study is interesting, I have thought that some time spent now in their consideration will not be lost. I would premise, in the second place, that in this paper I propose to consider the subject in its clinical aspects only.

Among the earliest to draw attention to tubercular neuritis were Joffroy (1), Eisenlohr (2), and Oppenheim (3), but the first exhaustive account with which I am acquainted is that by Pitres and Vaillard (4). These observers detail a number of cases, giving a very complete account of the symptoms, and as complete an account of the nerve changes found on subsequent histological examination. As regards symptoms, they allude to their great variety and diversity, a condition which can be understood when it is remembered that any part of the nerve tissue may be involved. They describe changes not only in the ordinary spinal, motor, sensory, or mixed nerves, but in the cranial, pneumogastric, and phrenics as well. They describe as symptoms not only sensory motor disturbances, muscular atrophies, and changes in the reflexes, but neuralgias, especially sciatica and trophic disturbances, especially herpes zoster. A very important feature which they refer to in connection with the symptoms is their mobility, their tendency to disappear, reappear, and alter. So marked indeed is this tendency, that

we can easily excuse the older clinicians for regarding them as accidental, and the more modern for ascribing them to vaso-motor changes.

As to morbid anatomical changes, they, like most of the writers who had preceded them, found that these seemed to be limited to the peripheral nerves, and that the large cord cells seemed unaffected. They allude to Eisenlohr's description of vacuolation in the cord cells, but they consider this change too indefinite to warrant the idea that there is any central lesion. They also quote instances of latent tubercular neuritis, *i.e.*, of patients who, with the exception of vague pains, showed no symptoms of neuritis, and yet in whom this pathological condition was found on *post-mortem* examination.

The subject of tubercular neuritis has been discussed subsequently by many observers—Suckling (5), Jaffa (6), &c., and it has been referred to in the various text-books on nervous diseases (7). Its importance, however, warrants further consideration, and the narration of certain examples of it may possibly have the effect of stimulating further inquiry.

The first case which I shall quote is an example of perhaps the commonest form of tubercular neuritis, *i.e.*, one presenting the ordinary sensory motor, reflex, and trophic disturbances.

CASE I.—J. S., aged forty, a sawyer, was admitted to the Infirmary on March 21st, 1895, complaining of cough, spit, and hæmoptysis. He stated that the hæmoptysis had come on seven weeks previously, but that he had had a cough since the previous June.

His family history was very good, he had always had plenty of good food, and he had always been moderate in the use of alcohol. His home surroundings had been good, but at work he had been much exposed to draughts. He had no previous illnesses, but eight years previously he had sustained a fracture of the left leg.

His present illness began in June last. He had been exerting himself at work, when he felt a sharp pain in his right side. This pain was worse on breathing, and was associated with a cough. He had to go home, and was off work for about a week. The cough lasted some time longer, but he went back to his work, and it got better. During the last two or three months, however, he had been feeling weak, though he states

that he has no cough. Then, seven weeks ago, he had a sudden severe hæmoptysis. Since then he has been feeling weaker, his cough has troubled him, and he has been complaining of prickling feelings in both legs and feet.

*State on admission.*—Height, 5 ft. 6½ in.; weight, 9 st., used to weigh 10 st. 11 lb.; pulse usually 112; temp. varies from 98 to 101.

*Alimentary system.*—Appetite poor, some thirst, occasional discomfort and heartburn after food, bowels constipated. On examination the abdominal organs were found all apparently healthy, but there was slight general tenderness on pressure all over the abdomen.

Circulatory system shows no evidence of disease. Pulse about 112, rather soft in character.

*Respiratory system.*—Respirations 28 per minute. The cough is frequent, and great difficulty is experienced in bringing up the expectoration. Sputum muco-purulent, nummulated, shows many tubercle bacilli. No hæmoptysis since admission. On examination marked dulness is found over both upper lobes, more extensively on the right side. Over the right upper lobe the breathing is cavernous, with coarse consonating crepitations. Over the left upper lobe the cavernous character is not so marked, but there are plenty of crepitations. All over the chest, especially posteriorly, the breathing is harsh, and crepitations and rhonchi can be heard.

*Urinary system.*—No pain or trouble with micturition. Urine 40 oz. acid; no abnormal constituents.

*Integumentary system.*—Skin soft, and patient perspires a great deal.

*Nervous system.*—Special senses normal. Skin sensibility to touch, pain, and temperature slightly impaired over both legs and feet. At those parts the patient has been feeling formication for some weeks past. Power of movement of the feet at the ankle joints is impaired, especially on the left side. Muscular wasting in the legs is marked, and there is some tenderness on pressure of the muscles.

This patient rapidly became worse, and with the aggravation of the chest symptoms, the indications of neuritis became more marked. On May 10th the entry is as follows:—"Patient much weaker, cough more aggravated and frequent, prevents sleep at night and brings on vomiting during the day. Eyes

sunken, cheeks hollow, sweating is profuse. On both legs, especially the left, the condition of drop-foot is present, and power of extension is completely gone. He complains much of cramps in the muscles of the calf, and on grasping them pain is experienced. There is also some pain when the muscles of the thigh are grasped. The reflexes—plantar, achilles tendon, and patellar tendon—are absent. With electricity the reaction of the muscles, both to the foradic and galvanic current, appears diminished. Skin sensibility to touch, pain, and temperature is lessened, but the sensory disturbances do not seem to be so great as do the motor." This patient died on May 25th. The condition of the legs and feet remained unchanged. For the last few days of his life he suffered from occasional very severe dyspnœic attacks, during which his pulse was very feeble, and his respirations rose to about 50 per minute.

The second case which I quote is one which illustrates, first, that the sensory tracts may be the more affected, and, secondly, the great mobility of the symptoms, *i.e.*, their tendency to disappear and reappear.

CASE II.—William M., aged nineteen, a porter, was admitted to Ward XXXI., April 22, 1898. He had lupus of the face of some ten years' duration, tubercular disease of the right tarsus of three years' duration, laryngeal and pulmonary tubercle of two years' duration, and tubercular bone disease in the right little finger of some months' duration. His family history was bad, and his surroundings during practically the whole of his life had been of a very miserable character.

*State on admission.*—Height, 5 ft. 7 in.; weight, 6 st. 7 lbs.; pulse usually about 100 per minute ; temp. varying between 97 and 100° F. He has a cough, with muco-purulent expectoration, showing bacilli, and both apices, especially the left, show dulness and crepitation. But the condition of the larynx was on admission the most important feature, for, owing to thickening and swelling there, he presented stridor and great dyspnœa. On April 28 the dyspnœa was so great that tracheotomy had to be performed. This relieved him very much, and although next day he had two fits, epileptic in nature, he steadily improved, so that, towards the end of May, he was able to sit up on a chair for several hours each day.

On June 2 the following entry occurs in the case-book :—
" Patient states that when he awoke this morning he noticed

that he had no feeling in the right arm and leg, and to some extent on the right side of the face also. On examination loss of sensibility to touch and pain is found over the right arm and leg, and to a less extent over the right side of the face. The skin of the trunk is not affected. There is no evident loss of motor power, no muscular tenderness, and no changes in the reflexes and electric irritability of the right lower limb as compared with the left. This condition persisted for three days, and then began to pass away, so that at the end of a week it was quite gone. The patient gradually improved in condition, and on August 4 he was able to go to the Convalescent Hospital. There, however, he did not improve, and on August 28 he returned to us, when the following was the entry of his condition:—

"Right arm and leg feel somewhat numb, also right side of trunk, and right side of face, but to a less extent. Sensibility to touch and temperature slightly impaired over those parts. He says he feels it rather sore when the right side of his head is being combed. Muscles of right arm and leg slightly tender and wasted and motor power impaired. Circumference of right forearm, 7½ in., left, 8 in., right thigh, 11 in., left thigh, 12 in. Muscles of right shoulder appear wasted, specially the supraspinatus. Knee jerks increased, the right specially so, and the right knee shows clonus slightly. Biceps jerk increased on both sides, specially on the right arm. Triceps jerk is less on the right arm. Cremaster reflex not marked on either side, abdominal and epigastric are more marked on the left side."

In the next case, motion was mainly affected, but the duration of the paralysis was short. There was also bone disease.

Case III.—J. G., aged twenty-two, a miner, was admitted to Ward XXXI., July 30, 1898. His complaints were loss of power in the left arm, cough and spit, and he stated that the cough and spit had been troubling him for thirteen months, and the powerlessness for the last four months.

His family history shows a tendency to tubercular disease. His surroundings at home are quite favourable, at work as a miner he is exposed to damp. He has had no previous illnesses, except measles when a child.

His present illness seems to have begun thirteen months ago. He began then to feel less fit for work. He was easily

put out of breath, and he felt giddy occasionally. Then the cough started, and about ten months ago he spat blood. This hæmoptysis, sometimes in considerable amount, about half a teacupful at a time, has continued at intervals, and he has gradually been getting weaker.

Four months ago he noticed that his left wrist was stiff and painful, and since then he has observed that his arm has been getting gradually weaker, so that now he cannot move it at all.

*State on admission.*—Height, 5 ft. 9½ in., weight, 9 stone 10 lb.; used to weigh 11 stone 4 lb. He appears fairly healthy, although rather thin. His temperature varies from 97° to 99°, his pulse 80 per minute. His alimentary, hæmopoietic and circulatory systems are all normal.

*Respiratory system.*—Respirations about twenty per minute. Cough not very troublesome, except in the mornings. Sputum moderate in quantity, muco-purulent, and showing a large number of tubercle bacilli. No hæmoptysis at present. On examination marked dulness was found over the left lung apex, extending in front as low as the second interspace, behind as low as the spine of the scapula. Over this dull area the expiration is prolonged, the breathing bronchial, and accompanied by crackling crepitation. The vocal resonance is markedly increased. The right lung all over appears quite normal.

The urinary system is normal, but an increase in the amount of phosphates is noticeable.

The integumentary system appears normal, except that the skin is soft and moist, and he has sweats at night. The left radio-carpal joint is swollen and tender, and shows evidence of tubercular disease.

*Nervous system.*—Here the main point of interest is the condition of the left arm. On examination the whole limb is seen to be distinctly smaller than its fellow, the fore and upper arms being 1 in. less in circumference on the left side than on the right. The muscles of the hand, forearm, upper arm, the deltoid, supra and infra-spinatus and rhomboid are all considerably atrophied. He cannot raise the arm from the side, nor flex or extend at the elbow joint. He cannot extend the fingers, and he can only flex them to a very slight extent. On examination, sensation to touch, pain, and temperature seemed unimpaired, but when the muscles of the forearm are grasped tightly he complains of pain, especially over the extensors. On testing

the behaviour of the affected muscles to electricity, there was found slightly increased irritability to the faradic current. The response to the galvanic current was practically the same as on the sound side. On tapping the biceps and supinator longus, their reflex irritability seemed to be somewhat increased.

The left knee jerk was distinctly more marked than the right, and on the left limb an ankle jerk could be elicited. The cutaneous reflexes, plantars, cremasteric, abdominal and epigastric, are all well-marked and equal on both sides.

The nervous system was otherwise normal.

Owing to the condition of the lungs it was deemed inadvisable to interfere with the joint disease at this time. The forearm and hand were accordingly put in a splint, and the joint was treated by the De Beer's method simply. Great improvement followed, so that in the course of a few days he gained some power over the deltoid, biceps and triceps muscles.

On August 15 appears the following entry :—"Wrist joint swollen as before, but all the movements of the left arm and hand can be performed, though much less perfectly than on the right side. When the splint is on, he can raise the left arm above the shoulder, but without the splint he can't do this, the pain occurring in the wrist seeming to make him powerless. The muscles of the hand, forearm and upper arm, and the deltoid, supra and infra-spinatus, and the pectoralis major of the left side are all much wasted. There is no muscular tenderness. Skin sensibility of the left arm is not lessened ; indeed, over the inner part of the upper arm, the radial aspect of the forearm, and over the thumb, it seems somewhat increased. With the faradic and galvanic currents the affected muscles respond slightly less readily than those of the sound right side. The left knee jerk is still slightly increased, otherwise the superficial and deep reflexes appear unchanged."

Pitres and Vaillard quote a case of neuritis occurring in a man of twenty-three, who had tubercular disease of the right os calcis. The affected bone was scraped, and temporary improvement followed. Soon, however, the disease began to reappear, and the patient suffered from diffused general pains in the limb, and feeling of prickling and formication in the sole of the foot. The limb was then amputated, and the wound healed rapidly, but now acute pulmonary tubercular mischief

set in. At the same time the pains in the affected limb returned as severe as before, and soon involved also the left leg, associated with feelings of prickling, etc., in the sole. The motor power remained unimpaired. This patient died shortly afterwards, and an examination of the nerves showed peripheral neuritic changes in the originally affected limb, and to a less extent in the sound left limb as well. To this case one somewhat analogous has occurred in my own experience, with, however, a more favourable termination. I shall now quote it.

CASE IV.—J. R., a stable-boy, from Prestonpans, was admitted to the Infirmary, January 23, 1896, complaining of a sore left foot and weakness of the left leg, and stating that he had been ill for about five months. His family history and surroundings at home and at work were favourable. His present illness began suddenly five months ago. He was walking home from work one night, when he was seized with a pain in the left foot. He is sure that he never strained the foot, and though he had sustained a slight injury to it two months previously, he is sure that his foot was quite well up to that night. Next morning he found the foot swollen and painful when he tried to walk. He was treated as for a sprain, with rest, fomentations, liniments, etc., but all to no purpose. Although the swelling went down, the foot remained exquisitely painful, and he could only hobble about with the aid of two sticks. He came to the Infirmary, to the surgical waiting-room. There it was found at the time that there was no distinct evidence of bone disease. As, however, the left limb was distinctly smaller than the right, he was sent to my ward for further investigation.

*State on admission.*—Patient is a healthy-looking lad, 5 ft. 2 in. in height, weighing 7 st. 9 lbs. He hobbles about on two sticks, and his left foot is evidently very painful and tender. There is slight thickening over the inner side of the left tarsus. His pulse and temperature are normal, and so are all his systems except the nervous and locomotory.

*Nervous system.*—Sensation in the lower limbs normal. Voluntary motor power in the left thigh and leg muscles impaired, and the muscles are very soft and flabby. Circumference of left thigh, 16 in.; of right, 17¼ in.; of left leg, 10½ in.; of right, 11½ in. Skin reflexes well marked on both sides. Knee jerks and ankle jerks much exaggerated on both

sides, but specially on the left. Knee and ankle clonus present on both legs, specially the left. Faradic and galvanic irritability slightly lessened in both lower limbs. As the result mainly of rest, this patient improved somewhat at first, but as weeks went by it was evident that no real improvement was occurring. Further, the signs of tubercular disease in the left tarsus became more apparent. He was accordingly sent to the surgical hospital, and amputation was performed. Marked tubercular disease was found in the bones, but unfortunately the nerves were not examined histologically. Clinically, however, it is interesting to note that, as the result of the amputation, the increased reflex irritability of the legs became less and less marked. Six months after the amputation, knee and ankle clonus could no longer be elicited on the right limb, although the knee and ankle jerks were still increased. One year afterwards all that remained was a slightly increased knee jerk in the left limb, *i.e.*, on the limb on which the foot had been amputated. The patient otherwise was quite well.

This case offers some interesting problems for discussion. Granting, for example, that there was some neuritic process in the neighbourhood of the diseased bone, are we to suppose that the extremely irritable condition of both lower limbs and the wasting of the muscles of the left lower limb indicated any sclerotic or other organic change in the lumbar cord? Secondly, was the disturbed condition of the lumbar cord due to the action of a tubercular toxin, was it simply due to the severe peripheral irritation, or was it due to a mixture of these? To the latter of these queries I would answer that the combination of the toxin with the peripheral irritation was the probable factor; to the former query I would answer that even though our newest histological methods might not have revealed much in the way of change, yet there is little doubt that the condition of the cord itself must have been a morbid one.

This latter consideration leads directly to the question of another set of morbid phenomena which, I think, may well be grouped under the head of tubercular neuritis. This is the so-called "causeless hemiplegia" which may occur in the tubercular. In this connection it is always to be remembered that in a patient with tubercular lung disease, tubercular pleurisy, etc., specially in an adult patient, the supervention of a tubercular

meningitis may be manifested by very anomalous symptoms. A hemiplegia coming on suddenly, and persisting, or after a day or two disappearing, is one of these. In such a case, when we find a meningitis on *post-mortem* examination, we ascribe the hemiplegia to the meningitis. But there are many cases of such hemiplegias in which, on *post-mortem* examination, we find no meningitis, nor anything else to account for the hemiplegia. Attention to such cases has been drawn by Dr Philip (8), and by him and by others, something of the nature of an œdema, or of a vaso-motor change, has been suggested as the probable cause. I think, however, we should rather regard it as the result of the action of a tubercular toxin, and if it be asked why this toxin should manifest itself in connection with a certain tract of the cerebro-spinal axis only, I would reply, that this is exactly what this toxin tends to do, and that though for this there must be some determining cause, we cannot necessarily detect it. In the ordinary tubercular meningitis of childhood, there is, I think, no doubt that this toxin must be acting on the nerve tissue, and causing nerve disturbances in addition to those caused by the inflammation itself. It is probable that the convulsive attacks and temporary paralysis in tubercular meningitis are largely due to the toxin.

But this consideration in its turn leads to the consideration of another class of cases, examples of which we meet with every now and again, viz., of patients in whom little in the way of symptoms have been complained of or observed, and who somewhat suddenly develop severe brain symptoms, convulsions or coma, and dying in a few hours, show, on *post-mortem*, evidence of the presence of tubercle, but no evidence of brain, heart, or kidney disease. Such cases might be very important medico-legally, and I have met with several examples of them. The following is the last of which I have had experience.

CASE V.—J. G., an adult male patient, aged about fifty, was brought to the waiting-room of the Infirmary at 10.30 P.M. on July 16th, 1898, by the police, who stated that he had been found lying on a seat in the Meadows in an unconscious condition. No drug, nor anything of a suspicious nature, was found on or near him. His fellow-lodgers afterwards told us that he had for some time suffered

from a cough, but that that day he seemed no worse than usual.

On examination he appeared to be unconscious, but when shaken up, he gave correctly his name and address. He also stated that he had swallowed no poison, and there was no smell to be detected in his breath. The pupils were small and the eyes divergent, and there was no spasticity of the arms and legs. The pulse was very rapid and feeble, the breathing was regular, and auscultation of the chest revealed all over rhonchi and rales. Temperature in axilla 99° F.

He was put to bed in hot blankets, and, for the collapse, a little whisky was administered. Immediately thereafter he took a kind of fit, the muscles of the arms and back of the neck being in a state of tonic spasm. The legs were unaffected, and the eyes did not move. He remained in this condition for about a minute, and then relapsed into his former state. After this attack, the pulse was very much weaker, and stimulants, ether, strophanthus, and brandy, were administered. This improved the pulse, but three more fits of the same kind followed within the next half-hour. By 11.30 the breathing had become stertorous and intermittent. He took several deep inspirations quickly, and then there was a pause, sometimes as long as for eighty seconds. A little urine drawn off with the catheter had a sp. g. of 1020, and showed neither albumen, blood, nor sugar. Rapidly the patient became worse, the pulse more rapid and feeble, the respirations more irregular, and the tracheal rattles more marked. Death took place at 1.30 A.M.

*Post-mortem Report.*—Body somewhat emaciated, some brown pigmentation of skin of trunk, old cicatrices in front of legs.

Brain.—Convolutions not flattened, superficial veins distended, with some hæmorrhages round them. Slight subarachnoid hæmorrhages on vertex of left side. Slight excess of fluid in ventricles, brain tissue very œdematous. Grey matter irregularly congested, specially in basal ganglia, and pons and medulla.

Lungs, left.—Upper lobe shows extensive grey tubercle, and early cavity formation at apex. Lower lobe studded with irregular areas of grey tubercle, with hypostatic pneumonia between.

Right lung.—Upper lobe shows signs of old pleurisy, and shows extensive grey tubercle, with diffuse pneumonic condition between. Middle and lower lobes show scattered grey tubercular areas, with congestion and œdema. Other organs appear normal.

I am well aware that in this case other possible causes of death might be suggested than an acute and rapid tubercular toxin poisoning. But this patient had no other disease than the tubercular lung mischief; he had left his lodgings that day much as usual, and he had become insensible and comatose in the course of a few hours, so that a diagnosis of cerebral hæmorrhage or some toxic nerve poisoning was arrived at. In this way I think we are entitled to look upon it as very probably an example of general nerve tissue poisoning by the tubercle toxin.

Lastly, I would suggest that the mental clouding and stupor, which supervene at the close of most cases of phthisis, and by which a good providence soothes the agony of the dying hours, may be ascribed to the action of this toxin, at any rate to a very considerable extent. The only other remark which I would make is, that in tubercular disease we are very likely to have toxic effects from other toxins than the tubercle. Apart from toxic resorption, from secondary organisms in tubercular cavities, etc., we know that poisons infective like malaria, or toxic like alcohol, are apt to assert their presence when a patient is weakened by tubercular disease. We know that when a patient who has had malaria in days gone by becomes phthisical, the malarial symptoms are apt to reassert themselves; and we know that when an alcoholic patient becomes phthisical, the supervention of an alcoholic neuritis is a frequent event. Still there is no doubt that a tubercular toxin is generated, and that it can produce more or less distinct effects on nervous tissue.

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## 2. ACUTE YELLOW ATROPHY OF THE LIVER

By David Robertson Dobie, M.D., C.M., Coldstream

Though the lessons which we have been taught during our student career, and later as practitioners, are numerous and important, one cannot help feeling that in very many instances we are lamentably ignorant of the workings of which the human body is composed, and we only profess to try to put the functions of the body right, if they have gone wrong.

Diseases of a complex and varied nature are constantly cropping up before our vision, impressing this fact very forcibly upon our minds. I am of opinion that the case which has recently passed safely through my hands emphasises the truth of this statement.

If ever in my life I felt in the position of a master of a ship with rudder and tackle gone, spars overboard, and nothing remaining but a frail wreck, with a valuable life to guide as best I could through a stormy sea, trusting more to the turn of the tide than to anything else, I did so through the early days of August last. I was face to face with a patient suffering from what I feel I am quite justified in diagnosing as acute yellow atrophy of the liver. This opinion will doubtless direct my reader's attention to text-books and authorities upon this most important and enticing subject, and in conjunction with the descriptions of the disease contained therein, I hope the case I am about to enter fully into and portray will be fully comprehended—symptoms carefully sifted and analysed in detail, and a correct estimate of the situation arrived at.

To the pathologist who makes the examination of the liver after death his special study, it seems impossible that any curative result can be brought about. He finds the liver cells in a state of demolition and disintegration, the red corpuscles of the blood diminished in amount and granular, the white corpuscles numerous; and all the excrement which the liver is supposed to destroy, in order to keep the blood in a condition of wholesome purity, is found in great abundance on crystallisation, namely, leucine and tyrosine, cholesterin and urea, in greater or less amount.

We are face to face with this complex and difficult question, Is the complex fluid called bile a secretion resulting from the metabolic activity of the protoplasm of the liver cells (Forster), or is it an excretion from the blood in the portal vein, through the agency of the liver by a process of filtration? It would be well to go back to our physiological text-books, and we cannot fail to recognise the fact that uncertainty does exist as to the origin of this most important fluid in the process of tissue formation and nutrition.

In all probability the pigments and biliary acids are formed in the liver, but the bile *per se* is only a more convenient arrangement of the animal economy, in facilitating the removal of the most poisonous and effete matters, which of necessity find their way into the blood.

The blood is continually changing as it proceeds on its course, leaving and taking up something at every new spot, and by those changes being always rendered more fit for the next duty it has to perform (Paget). We cannot fail to observe that the several substances which the blood contains separate themselves into groups for further use.

Each corpuscle is in fact a minute organism, within which chemical and vital changes take place, as John Hunter firmly believed.

I can arrive at no other conclusion in the case which I am shortly to bring before you, but that the symptoms were due to suppression of the secretion, not to any obstruction to the excretion of the bile; in fact, that this, like many other excretions in a normal state, is formed in the blood and eliminated at the liver, therefore accumulating within the vascular system, when its separation at the liver had been rendered impossible and therefore temporarily suspended.

Alison was the first to clearly demonstrate this fact, that the existence in the blood of matters destined to excretion in this, as in other organs, is probably to be regarded as a disease of the organ intended to carry out this function, although in point of fact the formation of such matters, in undue quantity or with morbid qualities, is really disorder of assimilation or of absorption. To each organ a peculiar and special power has been given of selecting out of the common storehouse those materials which suit it best to work with.

It is quite outside the purpose of this paper to enter into the vast and important subject of jaundice. We know that the symptom is the result of—

1. *Obstruction from catarrh of* (*a*) *the ductus communis choledochus*, (*b*) *ductus hepaticus*—not invariably in the smaller ducts within the gland at the very commencement of the small ducts at the circumference of the lobule. In cirrhosis of the liver, the cells of the lobule become deeply stained with pigment, though implication of the system does not become general until a much later period in the history of the affection.

2. *Obstruction from inspissated bile or gall stones in the cystic duct, or from long retention of bile within the gall bladder.*—The jaundice which follows the passing of a gall stone, such as I have described, is most frequently observed in first attacks, owing to the fact that the lumen of the duct must be subjected to intense pressure, with resulting inflammation of the mucous membrane and extension into the ductus hepaticus or the ductus communis. I have frequently observed patients who have had repeated attacks of biliary colic pass through the ordeal with little or no jaundice, owing doubtless to the fact that the duct had become permanently enlarged.

I am, however, convinced that we have jaundice arising from an entirely different cause, quite outside the liver and independent of any obvious change in the character or structure of that organ.

3. *Suppression of the secretion, not obstruction to the excretion of bile; the deviation from the ordinary course taking place within the circulatory area, from some cause as yet unexplained.* —Cases of jaundice under this, our third subdivision, are numerous and frequent. It would be positively absurd to eliminate the influence of mental emotion or severe nervous strain of an exciting or depressing nature, upon the secretion of bile.

When jaundice occurs suddenly under violent mental emotion, without any indication of calculi inflammation or organic disease, we are left to the conclusion that a reflex vasomotor influence from the central nervous system comes into play, and this fact has given rise to the popular belief, which exists in some countries, that excitation or depression

of the cerebro-spinal mechanism is a predominating factor in almost every case.

Certainly under this third head we include—(1) Jaundice from mental emotion; (2) jaundice from snake venom, certain fevers, mineral and other poisons.

To enter into details regarding all those agencies bringing about an attack of jaundice would interfere with the purport of this paper—for my patient's history entirely excludes any possibility of his having been exposed to any external cause, such as I have tabulated; but there is one influence not before taken into account, and apt to be disregarded in this country, and that is the very high and long-continued high atmospheric temperature prevailing this summer over the greater portion of the British Islands.

The meteorological statistics at the beginning of the month show that the mean temperature was far above the average in all parts of the kingdom, 78° as the absolute maximum being registered—the temperature of the warmest day and the warmest night being the highest registered in August since 1893.

During the early days of the month, this district was under the influence of a large anticyclone with a steady high temperature.

Leaving the speculative opinion as to the *fons et origo mali*, I now take the opportunity of bringing the case before you.

J. F. S., æt. 50, married, farmer. Birthplace, Bergedorff, Prussia, living now in Berwickshire, and in this country thirty-three years. A singularly healthy active man of small stature, with three children—one dead of scarlet fever, to whom allusion must be made, namely, that this little patient had malignant scarlatina, and before death the whole surface of the body was covered with purpuric spots.

HISTORY.—On the 1st of August the patient was in the very best of health and spirits, and actively engaged on his farm and in his garden. He was much heated—partly owing to the rapidity with which he goes about his business, and partly owing to the almost tropical heat prevailing in this country at the time.

He rested well during the night, breakfasted heartily, and started on the following day with his wife on a journey of ten miles, to visit an aged relative. The sun during the day

was very overpowering. On his return journey he felt decidedly ill; notwithstanding the external warmth he shivered from head to foot. The rigors were remarkable from their intensity. There was severe headache and general depression. However, Mr S. was just the man to discountenance any medical interference at such an early period of the disease, but, thinking this onset betokened a type of influenza, an immediate adjournment to bed was the only thing decided upon. I happened to see Mrs S. the following day on quite a different matter, but the somewhat sudden breakdown of her husband was recounted to me; and, acting on the information that he had been out of his usual the previous day, and had partaken of something which had not agreed with him, I ordered a seidlitz powder to be given immediately, and a dose of phenacetin should the intense headache continue.

*4th August.*—The patient has not yet been seen professionally, but I am told that he had spent a most miserable day—the great sickness and vomiting being uncontrollable.

*5th August.*—Early in the forenoon I was called for the first time, and I found all the symptoms of the day previous were exaggerated. The pulse was quick, the temperature 102°.8, and considerable tenderness existed over the whole abdomen, which I attributed to the excessive vomiting and retching. I made a careful examination of all the organs, and could not detect any abnormal condition in heart, liver, lungs, or spleen.

The urine was, however, highly concentrated, with about one-eighth albumin—no sugar. The specific gravity was 1022. I prescribed ice, Valentine's meat-juice, brandy, skimmed milk and weak tea. Medicinally I ordered mist. pepsin and bismuth, also a sharp purgative of calomel and colocynth, to be followed by a seidlitz powder.

*6th August.*—I found the patient not much changed for the better or worse; there was noticed a marked redness and flushing of the skin; also a most peculiar hue all over the face, the ears burning hot, the lips dry and parched. The temperature was 103°, and the urine was loaded with albumin and a very decided taint of bile-colouring noted. There was tenderness on percussion over the liver, but no enlargement or diminution in the size of that organ. There was great tenderness over the stomach, and an absolute inability to

swallow or retain even a tablespoonful of milk. The only medicament that would be received by the stomach was brandy and ice.

*7th August.*—I saw my patient in the afternoon, and found a certain improvement in the condition of his organs generally. The redness of the skin had given place to an intense blackening of the deepest jaundice type; the conjunctivæ resembled more those of a West African negro; the bowels acted naturally, but the exhaustion was extreme. I found the temperature 101°.4, and pulse 100, very weak and compressible.

*8th August.*—There was a decided fall in the pulse from 100 to 60, the temperature had gone down to 100°. There was complete drying up of all the salivary and mucous secretions about the mouth and pharynx. The jaundice was of a most remarkable type; it had more the appearance of a blackening, with a mummification or drying up of the whole surface of the body. The bowels were moved with hot Apenta water, and the sipping of this saline liquid was the only thing that relieved the intense thirst. It is worthy of note that there was perfect sensation, and the brain remained clear during the severe strain.

*9th August.*—The temperature had fallen to 99°.4, but the pulse still remained at 60, and was extremely thready and intermittent. The jaundice was intensified, the mouth and tongue having become quite black. Brain kept clear and calm, but the voice weak. The most alarming symptom, however, which manifested itself, was the severe hæmorrhage from the mouth and nose. There was constant spitting of dark, clotted blood. The treatment employed to combat this condition consisted of an astringent mouth wash and nasal douche. I changed the mixture to an acid, and gave a very grave prognosis.

*10th August.*—There were abundant hæmorrhagic petechiæ all over the forearms and upper part of the chest. The urine was found to be very black, like porter, and with great difficulty could it be passed on account of the long clots of blood. The urine on standing deposited blood in large amount. In the cylindrical vessel there was fully three-fourths of a deposit, which was microscopically examined.

*Examination of the urine.*—The transparent urine was of a very dark colour, having a specific gravity of 1024. Acid in

reaction, distinctly albuminous, without a trace of sugar, it presented all the characteristics of urine loaded with bile. Urea was measured by means of the simple glass tube and pipette; the former being filled with the standardised solution of hypobromite of soda, and the measured quantity of urine introduced. My estimate of the amount of the urea came to very nearly 3 per cent. of the specimen examined. For clinical purposes the result was quite satisfactory, but for precise physiological inquiry it was doubtless inaccurate. The microscopic examination revealed a mass of a very heterogeneous nature. The crystals largely predominated, and no difficulty was experienced in at once differentiating the rare forms of leucine and tyrosine. The spheres of leucine, so familiar to all microscopists, and the sheaf-like collections of tyrosine needles were very beautiful. Abundant evidence of hæmorrhage from the mucous lining of the urinary tract was displayed. Cholesterin was not even wanting in the field of the microscope. I was struck with the beauty of crystallisation. What purity, combined with such a lethal poison, existed there. Surely those crystals had cast out all impurity; but they were themselves foreign to the elements they moved in, therefore their presence indicated a chemical disorder with a potency capable of paralysing animal life and energy.

11*th August.*—I found the patient very poorly, and in a weak, exhausted condition. The amount of bile and blood in the urine had markedly increased. From the bowel there was passed abundant quantities of mucus mixed with blood. The body was covered with petechiæ, more particularly over the abdomen. From the mucous surfaces and from the lips and gums there exuded blood and mucus, truly a most pitiable object to behold. The examination of the liver disclosed the fact that the area of dulness had really disappeared. I visited the patient at night, hardly expecting him to live until next morning; but in order to insure a peaceful night I gave a large dose of bromidia, which, however, had little effect one way or other. I gave the opinion that it was only a matter of time, and my visit would be made next morning early, when I would endeavour to stop the hæmorrhage by the administration of a powerful astringent, which I would bring with me.

12*th August.*—I was early on the road to visit the patient, when I met one of the servants hurrying to tell me that Mr S.

was dying—"to come quickly." I was not surprised to hear it. As I expected, I found the patient very weak and exhausted. His appearance was remarkable. The condition already described was aggravated, and yet his brain remained quite clear, and on my urging him to make all things ready for his departure to the unknown, he willingly and bravely did so like a hero. Everything had been given him except the turpentine, which I had with me. I gave him about a teaspoonful of the oil of turpentine in a mucilaginous mixture. It was retained, as everything else was at this stage. The same quantity was retained in the afternoon, four hours after the first. On my visit in the afternoon I found a change had come over the patient. He felt better and had a cheerier look. He seemed to experience a new life, and had strong hope of yet being able to pull through. There was no sickness. His pulse felt fuller and stronger than it did in the morning. The petechiæ were very numerous all over the body, but the bleeding from the mucous membrane of the mouth and throat had ceased. I remained all night, giving at intervals of four hours about 15 minims of the mixture. In the early hours of the morning sleep returned, and on waking about 6 A.M. I gave a large dose of Apenta water. The bowels were slow to act, so I gave an enema and waited the result, which in the end was satisfactory, the motion smelling very strongly of turpentine. I had already asked my colleague to come and see the case with me, but he was unable to do so until the morning of the 13th. I pointed out all the important points and features in the case, and he can give an independent testimony of the condition, which he looked upon as very hopeless. The condition of the liver was fully recognised, but a change from the day previous had come about. There was dulness where before none could be detected. A finger's-breadth, however, would cover the area in the vertical direction. I was fortunate in obtaining the services of a professional nurse from Edinburgh, who took the greatest possible interest in the case, which to her and to me was unique. Her notes are herewith appended.

"14*th August.*—Restless night, but on the whole a number of favourable symptoms have presented themselves. Slept at intervals, and takes nourishment. Urine is passed more easily and is clearer, but intensely coloured with bile. The albumin

is gone. The simple administration of 1 gr. of calomel at bedtime, followed by a dose of Apenta water, brought about a copious evacuation of the bowels.

"*16th and 17th August.*—Improvement continues uninterrupted, but the number of petechiæ over the abdomen and thorax has markedly increased."

On my visit on the 17th I noticed a marked increase of liver dulness; fully 2 in. could, without any difficulty, be easily made out.

"*18th August.*—The patient expresses himself as being fifty per cent. better. He has now a moist, cleaning tongue, and no bad taste in the mouth. The urine is cleaning rapidly. The ears are assuming a natural reddish hue; the colour between the fingers is coming round to the natural. The sleep which he now gets is refreshing and prolonged."

From this date onwards up to the present time there was a clearing away of all the symptoms. Nurse had left after a fortnight's untiring attention. The patient was now very yellow, combined with a certain amount of paleness. On 2nd September, when the last note was taken, we find the patient in his chair at the bedside, with a brisk appetite, regular motions, under a regimen of milk, fish, porridge, tea, coffee, mutton or chicken-soup. Brandy had been administered in large doses up to the present, but the need for any more than one teaspoonful at a meal or after any exertion was quite unnecessary. No further notes were taken.

It is awful to think that peril is so imminent in the midst of apparent health.

"Sub sole sereno—nubem,
Sub risu—lachrymas";

though in truth, if we could look into the innermost structures of all the vital organs, we should often have to notice seeming security connected with the utmost hazard.

Remarks.—The mode of onset and the character of the symptoms in this painfully interesting case, its progress and its termination, are quite consistent with the belief, doubtless a true one, that this was a case of acute yellow atrophy. The cure of a case like it is so very rare, that doubts must arise in the minds of many as to the accuracy of such a diagnosis. It is only in the *post-mortem* room that our opinions are verified

or negatived, and if a fatal result had come about I would have carried out a careful examination of all the organs, and proved the correctness, or otherwise, of the diagnosis. My colleague fully concurred in the gravity of the disease, and we were quite at one as to its ultimate issue.

The subject is such a complex and interesting one, that my paper would be incomplete without a few practical and theoretical considerations.

First, let us consider shortly the normal situation and size of the organ. The liver is situated in the right hypochondrium, filling up the concavity of the diaphragm, and protected by the ribs, leaving only a small portion of the left lobe uncovered, which projects into the epigastrium. It is not difficult, by means of percussion and palpation, to recognise the organ, but certain impediments exist which must be overcome in order to arrive at an exact determination of its dimensions. The true upper border lies in the fifth right intercostal space, and slopes from before backwards to the vertebral column, where we find it in contact with the ninth and tenth intercostal space. The whole upper border projects beyond the middle line for about $2\frac{1}{2}$ to 3 in., until the dulness elicited by percussion merges with the apex of the cardiac dulness on the left side. The position of the lower border varies so much in different individuals that it is hazardous to express any definite opinion in regard to it. Tight-lacing, abnormalities in the chest wall due to spinal caries, rickets, or the accumulation of fluid in the chest, have a depressing effect on the situation, but it may be fairly assumed that the lower border extends for 3 in. beyond the edge of the thorax—though in many cases it is much lower. When the extension or protrusion of the lower border is much beyond the boundary, the organ may fairly be assumed to be enlarged or dislocated. In this case we had marked increase in the volume of the liver on the day I first saw the patient, and I am sure, from the history of the great discomfort and rapidity of breathing during the onset, there had been tumefaction to a great extent. During the three subsequent days there was rapid diminution, until the Friday, when a fatal termination seemed inevitable—the liver dulness had disappeared entirely. The organ seemed to have receded to the vertebral column. When my colleague examined on

Saturday morning, the dulness could be made out to be about a finger's-breadth, and I remarked to myself at the time that there was now a dull percussion note where before there had been absolute resonance. On subsequent examinations the organ was found to be gradually regaining size, and now the lower border can be quite distinctly felt in its normal position.

*Urinary organs.*—The quality of the urine was normal in amount during the first week, but when the hæmorrhage was severe it was diminished in volume and altered in consistence. I have already entered into a detailed description of the pathological appearances. As time passed on, the deposits cleared away, and nothing now remains but a greenish coloration. There is no albumin in the urine at the present time.

*Nervous system.*—Beyond marked typhoid prostration on the ninth day from the onset of the illness, there was nothing to note. Herein does the parallel with other reported cases of acute yellow atrophy of the liver fall to the ground. The patient's mind remained clear throughout. He was all along able to recognise those around him, and he was perfectly calm even when life seemed to be ebbing away. The pupils remained in a normal condition ; there never was xanthopia, or yellow vision ; sensations were unimpaired, and there was no itching of the skin.

I must now, with considerable diffidence, shortly enter upon the most difficult part of the subject, namely, to account for all this disturbance of the physiological functions connected with the digestive mechanism. Can no definite theory be advanced to clear up the mystery ? The liver, or factory organ, gets the blame. My opinion from clinical observation is, that this is a purely blood disorder, with such an intense vasomotor change within the liver that total collapse of the organ is the consequence.

Why do we meet with the symptoms I have described in so many conditions of blood poisoning ? I have enumerated them. In such cases we have a distinct cause and effect ; but here in this case we are bound to exclude all these causes, with the exception of the abnormal high temperature of the atmospheric air, with extreme dryness. That there was an influence of the nature of a miasm or contagium vivum contributing to the production of the disease, I am inclined to believe, but the

exact bacteriological condition within the vascular area I am not prepared to state. There was some activity amongst the white leucocytes, and enormous destruction of red corpuscles, setting loose all the elements resulting from the digestion of nitrogenous tissue. The metabolic activity of the liver cells became disorganised and paralysed, finally disintegrated, and annihilated. This pathological condition is found invariably in those numerous fatal cases, and would have been found in my case also, had a fatal termination been the result, as was looked for and expected. The theoretical consideration of this obscure and rare complaint must be left in the hands of abler and more scientific observers than myself.

If I have succeeded in arousing in the minds of practitioners a fact that this disease is, as I believe, not a primary liver affection, and that it admits of a cure by a simple remedy, —for I have only to thank the one remedy employed, and which I do not notice ever to have been tried before,—then my labour in bringing this subject before the profession has not been in vain.

I cannot think that the *Lancet's* advice to students should be overlooked even by practitioners of many years' standing—

"All who follow the medical profession must be students for ever; from every case the real physician and the real surgeon has a lesson to be drawn. No man can dispense with observation or the results of observation. This need is never finite. For book learning there is a substitute, namely, experience; for experience there is no substitute."

Finally, in dealing with the treatment of this condition, I must subdivide the subject into three heads—(1) Treatment of the premonitory symptoms; (2) treatment of the hæmorrhagic stage; (3) treatment of convalescence.

1. *The treatment of the premonitory symptoms* resolves itself into a very simple matter, and anything of a palliative nature to relieve the nausea, sickness, vomiting and thirst, must be appreciated by the intensely congested mucous lining of the digestive tract.

2. *The treatment of the hæmorrhagic state* is the most serious duty we have to perform. We know the dangers of delay in a stage like this, and we must guard against the loss of the living corpuscles amongst the dead. I have only to thank the

administration of the oil of turpentine for saving the life of this patient.

Its actions and uses are manifold; as an antiseptic it is in daily use, and in small doses internally, such as 15 to 20 minims, it quickens the pulse, raises the animal heat, and even produces exhilaration. Now this is just what actually did take place. The patient felt the medicine "go through him."

When this action is steadily kept up, as it was in the case before us, the drug acts as a tonic of a very powerful kind, and is therefore useful in the typhoid condition of continued and other fevers.

Like many other volatile oils, it has a specific action on mucous membranes; its permeability through all excreting organs is undoubted, and although I cannot claim for it a specific property in jaundice, I am quite justified in placing the oil of turpentine amongst the most valuable thereapeutic agents we possess.

I attach much importance to the careful attention of the "primæ viæ," by the regular administration of 1 gr. of calomel every night at bedtime, followed by a saline draught, in the shape of Apenta, next morning.

3. *The stage of convalescence is protracted and tedious.*—Careful dieting at the hands of a trained nurse is of the utmost importance. Fresh air and wholesome water are indispensable. Under such a course the improvement in colour of the skin and restoration of the wasted muscles had remained up to the present uninterrupted in my patient. Relaxation from anxiety and gentle country exercise will very speedily effect a complete cure.

The question now before us all is, What about the condition of the liver now?

A week ago I saw my patient at the sea-coast, and, after careful examination into every detail, I must conclude that the organ in structure and function seemed as good to-day as it was before he took ill two months ago.

## Meeting II.—December 7, 1898

SIR JOHN BATTY TUKE, *President, in the Chair*

### I. ELECTION OF MEMBERS

J. Taylor Grant, M.D., B.Sc., and Neisch Park Watt, M.B., were elected Ordinary Members of the Society.

### II. EXHIBITION OF PATIENTS

1. *Dr Harry Rainy* exhibited a case of MUSCULAR DYSTROPHY. The patient, aged 23, had for the last eight years followed the trade of a painter, being chiefly employed in washing and size-colouring, in the course of which he often had to remain for many hours in wet clothes. During the nine months that his illness had lasted the most important symptom had been a gradually increasing weakness of both arms, with stiffness and pain in the shoulder and elbow joints. Latterly these conditions had become so pronounced that he could no longer follow his occupation. An examination of the patient revealed marked wasting of the triceps, deltoid, and lower part of the pectoralis major on both sides, whilst the supraspinati and infraspinati muscles were also impaired in a less degree. The rest of the muscles of the arms and hands were, on the contrary, quite unaffected. No reaction of degeneration was present in any of the affected muscles; fibrillary twitching and myotatic irritability were also absent. The arms were kept in a semi-flexed position, any attempt to straighten them causing the patient great pain. These symptoms seemed to exclude the possibility of the disease arising from any lesion in the spinal cord; leaving a diagnosis to be made between a muscular wasting consecutive to rheumatic inflammation in the joints, and a primary myopathy. The latter alternative seemed to be favoured by the well-marked and selective bilateral symmetry of the condition.

2. *Mr H. J. Stiles* exhibited a child six months after reduction of a CONGENITAL DISLOCATION OF THE HIP by the method of Lorenz. (This patient was exhibited to the Society last Session, see *Transactions*, vol. xvii., p. 108.) The

patient, a girl aged four months, was exhibited to the Society a little more than six months ago, when Mr Stiles stated that the case was a suitable one for treatment by the bloodless method of Lorenz—a method which had been practised with great success on the Continent, but which had not been taken sufficient advantage of in this country. In this case, the first attempt at reduction failed, due, apparently to an insufficient amount of force having been applied. The patient was put back to bed, and a weight of twelve pounds was applied to the limb for six weeks in order to stretch the shortened muscles. The second manipulation, which was successful, was carried out as follows :—Chloroform having been administered, the child was placed upon a mattress on the floor and manual extension made by grasping the limb above the knee, an assistant keeping up counter extension by means of a skein of worsted looped round the perineum. When, by gradually increased extension, the trochanter had been brought down to its normal level, the thigh was flexed to a right angle with the trunk and rotated slightly inwards, extension being maintained. The next step consisted in slowly abducting the limb until it reached the horizontal plane, and projected outwards at right angles to the trunk. This must be done gradually and with care to avoid fracturing the neck of the femur. When the above degree of abduction was reached a distinct reduction impact and sound was felt and heard, and the thigh then maintained spontaneously the above characteristic position of right-angled abduction. Any attempt at abduction results in relaxation. The reduced limb was then put up in plaster (strengthened with strips of aluminium) in its abducted position, extending from above the pelvis to a little above the knee. After six weeks the plaster was removed, and, the abduction having been slightly reduced, the bandage was reapplied. The child, who had previously been kept in bed, was now provided with a two-inch sole to the boot of the sound limb, and encouraged to walk about the ward as much as possible, in order that by throwing the weight of the trunk upon the affected limb the head of the femur (now occupying its normal position) might, by the pressure exerted upon the pelvis, deepen the rudimentary acetabulum. At the end of a second period of six weeks the amount of abduction was again reduced, and the plaster bandage reapplied. A

month later the abduction was still further reduced, and the child sent home with a one-inch sole to the boot of the sound side. The patient returned to hospital only a few days ago to have the plaster permanently removed. Examination of the limb showed that, with the exception of slight external rotation, there was now no deformity; there was no shortening; the trochanter occupied its normal position; the head of the femur could be felt rotating at the base of Scarpa's triangle; the folds of the groins and buttocks were symmetrical—in fact, there were now no signs of the original dislocation. Mr Stiles thought that the case illustrated very forcibly the value of Lorenz's method, and that it bore out all that he claimed for it. Every endeavour was made to follow accurately the directions given by Lorenz in a paper published in the *Transactions* of the American Orthopedic Association, vol. ix., 1896. For the anatomy of the condition and the rationale of the method, this paper should be consulted. Mr Stiles concluded by pointing out how important it was to diagnose congenital dislocation of the hip in early childhood, as, after five years of age, the bloodless method is seldom successful. The results of operative treatment were now known to be far less satisfactory than was at first claimed.

3. *Mr C. W. Cathcart* exhibited a case of GOITRE. Symptoms resembling those of myxoedema—rapid improvement after removal of tumour. (This case was recommended to Mr Caird, and sent on by him to Mr Cathcart.)

Mrs W., aged 53. Admitted to the side room of Ward 5 on the 10th of October 1898. She complained of the swelling in her neck, accompanied by great general weakness, and occasional severe attacks of dyspnœa.

*History.*—About twenty years ago, soon after the birth of her second child, she noticed a swelling in the front of the neck. This, however, disappeared in a month or two after being painted with iodine. It showed itself again seven or eight years ago, but for some years she had nothing done for it. In the spring of this year she was much run down by nursing a relative, and afterwards took influenza, after which her general health grew worse, and the swelling increased especially in the last few months.

The symptoms resembling those of myxoedema had come

on within the last two or three years, and were the following:—

*Features* had become broader, the skin flabby, and the wrinkles had been obliterated by swelling, especially under the eyes. Complexion dirty-looking. Expression dull and apathetic.

*Tongue* had latterly felt too large for her mouth, and her speech had become thick and indistinct, she thought, from this cause.

*Skin* had seemed to herself to be dry.

*Hair* she believed to have fallen out lately.

*Nails* had become very brittle.

*Hands* had become much thicker and more clumsy during the last few years. Her marriage-ring has had to be enlarged once or twice.

There were, however, several points in which the symptoms differed from those of myxoedema, viz.:—

*Skin*, although complained of as dry by the patient, did not feel particularly dry to the observer.

*Hair*, in spite of her own statement, was still thicker than in most women of her age.

*Nervous system.* Although she speaks slowly and thickly, her intellect does not seem to be really impaired, and instead of being apathetic, she was nervous and easily upset by disturbances in the ward.

*Pulse* was quick rather than slow.

*Hands* were thought by some to be more like those of acromegaly than of myxoedema. The Röntgen rays shows the bones to be somewhat enlarged.

*Treatment* with thyroid tablets had had only partial benefit. The growth had diminished while she was taking them, but she had lost flesh rapidly, and had suffered from an increase of vomiting.

Besides these symptoms, she had dyspnœa, sometimes very troublesome at night, and a certain amount of dysphagia from the pressure of the tumour. She was much troubled with neuralgia, and was also so weak that on admission she could scarcely stand.

*Operation* was performed under chloroform on the 16th of November with Mr Caird's assistance. Kocher's method was followed. The enlarged lobe was removed. The right

lobe was slightly enlarged also, but was not disturbed. The case presented no special difficulty. The wound healed by first intention.

*Improvement* since operation.

*Tongue* was felt to be less by the patient very shortly. A week after the operation she told us that she could speak more easily.

*Face* has become distinctly less swollen, and the wrinkles are now beginning to show themselves.

*Skin* has become more moist.

*Hands* she has noticed to be smaller; her ring is now looser than at the time of the operation.

*General health* greatly improved. She feels much stronger and brighter—better, every way, than she has been for a long time.

Symptoms due to the mechanical presence of the tumour have disappeared, *i.e.* dysphagia, dyspnœa.

On section the enlargement was apparently due to colloid accumulation in the gland follicles forming cysts here and there. In places the cyst walls were calcareous.

Under the microscope the follicles of the enlarged thyroid gland are seen to be much distended with colloid material.

## III. Exhibition of Specimens

1. *Mr J. M. Cotterill* exhibited (*a*) THE TUMOUR, A GLIO-SARCOMA, which he had removed from the brain in Dr Russell's case. The tumour was the third which had been removed from this patient. In the first case the growth was about the size of a walnut, and was found to infiltrate the brain in its immediate neighbourhood in such a way as to make it necessary to cut away a margin of apparently healthy brain tissue in removal of the tumour. The patient improved very much after operation, as regards pain, speech, sight, and sensory and motor condition of the arm.

At the second operation (some thirteen months after the first operation) the patient had relapsed into a very similar condition as she was on the first occasion. Relief was again obtained for several months.

Upon the third occasion the tumour was the size of an orange, and projected considerably from the surface of the

head. The patient was in a very weak condition, and succumbed some three days after operation.

(*b*) THREE SARCOMATOUS TUMOURS removed from the brain of another patient, and pointed out how they varied from the glioma in the matter of encapsulation, the sarcomata being definitely encapsuled and shelling out with great ease.

2. *Mr H. J. Stiles* exhibited the FRACTURED FEMUR, HUMURUS, AND CLAVICLE OF AN INFANT, which had been brought to the Children's Hospital a few days previously in a shockingly emaciated and neglected condition. The history given was that, owing to ill-health of the mother, the child had not been lifted out of bed for a fortnight. The child was syphilitic as well as very rickety, and there were deep bed-sores over the hips and sacrum. Both clavicles, both humeri, and both femora showed more or less recent fractures of their shafts, with shortening and angular deformity. Early general tuberculosis was revealed at the *Sectio*. Skiagrams were taken after death by Dr Rainy, and showed extremely well the exact nature of the various fractures, some being of the green-stick variety, others with marked displacement, but in this case with complete tearing of the periosteum.

3. *Dr Scot Skirving* exhibited (*a*) EXTREME ENLARGEMENT OF ISCHIAL BURSA WITH PECULIAR CONTENTS. The patient, a sailor, had had a somewhat similar swelling on the opposite side six years ago, which was opened and the contents evacuated. While recovering from this he thought he used to sit almost entirely on the sound side. Soon afterwards the present swelling began to appear. It, however, grew very slowly, until a year ago, when it rapidly enlarged.

When examined the swelling was found to be the size of a large cocoanut, somewhat conical in shape, and the skin over the apex showing signs of giving way. Until incised it was diagnosed as an abscess. Its contents were bodies of a peculiar shape and about two ounces of synovial fluid. The cavity of the bursa easily admitted a closed fist, and the bursa itself extended upwards under the gluteal muscles and downwards under the hamstrings. Its walls averaged about a $\frac{1}{4}$ inch. in thickness, and in some places were much thicker. Over the tuber ischii the diameter of the wall was as much as an inch.

It was found impossible to dissect the bursa out entire owing to its adherence to surrounding structures, the great sciatic nerve amongst others. The bodies, which resembled gun wads more than anything else, were irregularly circular in shape, flattened, and of a dirty white colour. There were many hundreds of them, and they varied very considerably in size, from $\frac{5}{8}$ of an inch to $\frac{2}{3}$ of an inch in diameter. The thickest were about $\frac{1}{8}$ of an inch. Some were adherent to one another forming rouleaux, while others were still attached to the wall of the bursa. No tubercle bacilli was found either in the wall or in the bodies. The bodies consisted of fibrinous material. The bursa was completely extirpated, and the patient made a good recovery.

(*b*) RUPTURE OF KIDNEY AND LIVER. The patient, a very muscular man of 38 years, was admitted to Leith Hospital in September 1898, having fallen under the wheel of his cart in trying to jump up to the front seat. The cart was heavily laden, and was stopped by the bulk of the man. The wheel did not actually go over him, but pushed him on for several feet before the cart was brought to a standstill. Shortly after admission patient showed signs of considerable shock, but rallied remarkably under stimulants and rectal injections. This improvement was so well maintained for the next eight hours that the house surgeon did not send for assistance. After this, however, the pulse became very feeble, and the patient again showed signs of collapse. The urine when drawn off showed a very large admixture of blood, and a fracture of the eleventh rib was also made out. The normal area of liver dulness was much diminished. There was marked bulging of the right loin and extreme tenderness over the whole of that side. Rupture of the right kidney was diagnosed, and operation decided on. On turning the patient over, however, on to his sound side, he suddenly became unconscious, and, in spite of free stimulation, died almost immediately afterwards. The *post-mortem* examination showed the following:—Fracture of the eighth, ninth, tenth, and eleventh ribs; complete horizontal division of the right kidney, the upper two-thirds being separated by a clot from the lower one-third; enormous retroperitoneal extravasation—amounting to several pints—in the neighbourhood of the right kidney, and displacing the intestines upwards; much ecchymosis of both the large and small gut;

finally a rupture on the postero-superior aspect of the liver, 4 inches in length, and ¾ inch in depth at the deepest part. The interest in the case lay in the extraordinary vitality shown by the patient. He lived nine and a half hours after the accident, and was perfectly conscious throughout until within two or three minutes of his death. No doubt the rapid collapse at the end was due to fresh bleeding in quantity set up by the movement of the patient.

4. *Mr C. W. Cathcart* exhibited a specimen of STRANGULATED HERNIA. Excision of gangrenous bowl and immediate suture. Death from cancer of the stomach eight months afterwards.

Elizabeth C., aged about 55, admitted 15th February 1898, suffering from strangulated hernia. The symptoms had been present for two days, although at first they had been mild and somewhat obscure in character.

The operation was performed within an hour of her admission to the Infirmary. A Littré's hernia was found to be present, and the bowel which had been nipped in the hernia was so doubtful, that it was thought better to excise it altogether. A piece about 1½ inches long was taken away, and the ends immediately united with two rows of continuous stitches made with fine silk. She made a good, although slow, recovery, and left the Ward on the 6th of May. Her bowels gave her no trouble, but she had suffered a good deal from pain in the stomach and indigestion, and an obscure swelling in her epigastric region had been watched with considerable anxiety.

On the 7th July she was re-admitted on account of sickness and vomiting and increasing emaciation. A hard lump was now distinctly to be felt in the epigastric region. An exploratory incision was made, inoperable cancer of the stomach recognised, and the wound was immediately sewn up again. She grew gradually weaker, and died on the 9th October, *i.e.* nearly eight months after the operation for strangulated hernia.

The specimen shows that the double line of stitches has caused a ridge to form within the canal at the part farthest from the mesenteric attachment. The lumen of the gut has been somewhat diminished thereby, but it has been large

enough to permit the passage of food without any difficulty. From the time of the operation, up to her death, there was no symptom of obstruction. There were gastric disturbances, of course, for which the cancer of the stomach sufficiently accounted.

Under the microscope, the union is seen to be due merely to adhesion of the peritoneal surfaces of the bowel. The mucus membrane is continuous over the edge of the projecting ridge.

## IV. Original Communications

### 1. REGURGITATION AT THE PULMONARY ARTERY

By Francis D. Boyd, M.D., F.R.C.P.Ed., Physician to the Deaconess Hospital, Edinburgh

Regurgitation at the pulmonary orifice of the heart is a rare condition, and it is on account of its rarity and the difficulties which beset the physician in establishing the diagnosis that I venture to bring two cases under the notice of the Society to-night.

The cases represent well the two factors which may result in the establishment of the lesion apart from congenital causes. Firstly, endocarditis affecting the pulmonary valve cusps; and, secondly, increased pressure within the pulmonary circuit resulting in dilatation of the pulmonary artery and consequent incompetence of the valve.

Case I. Mary F., age twenty-five, residing at Uphall, was admitted under my care in Deaconess Hospital in October 1897 complaining of weakness and pain in the left side.

For some months the patient had felt out of sorts. Three months before admission she began to suffer from pain in the left lower costal region. The pain was of a dull character and was increased by fatigue. Up to the age of sixteen her health had been exceptionally good, but since then she had not been very strong though there had been no definite illness.

On admission the patient appeared a well-developed young woman. There were no obvious morbid appearances.

The circulatory system showed no subjective phenomena. The apex beat was in the fifth interspace in the mammary line, the cardiac pulsation was gentle and diffused. There was no

thrill. Percussion gave the right border of the heart 1¾ inches to the right of the mid-sternal line, the left border ¼ inch external to the mammary line. In the mitral area a soft blowing systolic murmur was heard which was not well conducted. In the tricuspid area a similar murmur was audible and the second sound was impure. Passing up along the left border of the sternum the second sound increased in loudness and the impurity became more marked, amounting to a slight murmur accompanying the second sound. This was best heard in the second left interspace 1 inch to the left of the sternal border. At the pulmonary area a soft blowing systolic murmur was audible. On passing to the right the impurity in the second sound could be traced on to the left half of the sternum, but at the aortic area the second sound was quite pure. Pulse 96 per minute, tension low, volume fair, vessel healthy.

The hæmopoietic system showed marked enlargement of the right cervical glands. No other glands enlarged. The spleen was easily palpable and tender. Anteriorly it reached to the mammary line and passed as low as the level of the umbilicus. Above the splenic dullness reached the seventh interspace.

The red corpuscles numbered 3,080,000 per cubic millemetre. The hæmoglobin was 40 per cent. There was an increase in the polynuclear leucocytes.

The respiratory, urinary and nervous systems were normal.

During the next two months the patient had several irregular rises of temperature, with rigour and increased pain in the spleen. By the end of a month the impurity of the second sound had increased to a definite murmur audible down the left border of the sternum.

Dr Gibson saw the patient with me, and as we considered the condition to be one of subacute septic endocarditis, I punctured the spleen for bacteriological examination. The examination, however, proved negative. As, however, the temperature continued to rise at irregular intervals, and the patient was getting worse, antistreptococcic serum injections were administered. The temperature after the injections improved decidedly. By the end of January the pain and tenderness in the splenic region had disappeared, and the general condition had improved. The condition of the heart

had changed. The right border was 1¾ inches to the right of the mid-sternal line, while the left was 1½ inches outside the mammary line. In the mitral area the systolic murmur was audible as before, and there was in addition a faint diastolic murmur. Both murmurs were audible in the axilla, the diastolic increasing greatly in loudness as the stethescope passed up towards the axilla. At the angle of the scapula no systolic murmur could be heard, but the diastolic was distinct. In the tricuspid area a normal first sound was followed by a loud diastolic murmur, which could be traced up the left side of the sternum to its point of differential maximum intensity in the second interspace 1 inch to the left side of the sternal border. The murmur was well heard upon the sternum, but was immediately lost on passing off the sternum to the right. In the aortic area both sounds were quite pure, the second being accentuated. The patient improved, and by the end of January was able to leave hospital and return home, where she remained, but was never able to do any work. In June she returned to hospital feeling worse. There was some oedema. The heart was more enlarged. The diastolic murmur had increased in intensity and duration. The urine contained albumen and tube casts. The liver was enlarged. After a time she improved slightly and returned home. There she gradually grew worse and died in September. As I was absent on the continent there was unfortunately no *post-mortem* examination.

The case is of considerable interest as it is seldom that we get an opportunity of watching the gradual development of this lesion. When first admitted the patient had signs of anæmia, with enlargement of the spleen and enlargement of the glands in the neck. Examination of the blood, however, excluded the diagnosis of splenic anæmia, Hodgkin's disease and leucocythæmia. The blood showed an increase in the polynuclear leucocytes—it was, in fact, the blood of an inflammatory condition, and this opinion was borne out by Dr Gulland, who kindly examined some films for me. Taking, then, into consideration the condition of the blood, the temperature, and the gradual increasing impurity in the second sound at the pulmonary area, there could be little doubt that the patient was suffering from subacute endocarditis of the pulmonary valve of a septic nature. Though the bacteriological

examination of the blood was negative, it was thought worth while to try antistreptococcic serum. The injections were followed by decided improvement in the temperature; the rigors and irregular rises of temperature ceased, and the general condition of the patient improved.

The second case forms an excellent contrast.

CASE II.—Christina C., age eighteen, single, was seen by me at the New Town Dispensary, and was admitted to Ward XXXIII., Royal Infirmary, under the care of Dr Wyllie, in October 1896, suffering from breathlessness, pain in the chest, and cough.

The patient had enjoyed good health till three years before she came under my care, when her legs, arms and face began to swell, and she had pains in the legs which were so severe as to prevent her from walking. She was ordered rest, and after five weeks she improved and was able to go about again. She remained in good health for two years, at the end of which time she began to suffer from shortness of breath, which was most marked on exertion, and from pain in the chest. At this time there seems to have been no swelling of the limbs. She was in hospital for three months, and when discharged was feeling much better, and continued well up to her present attack, when she complained of breathlessness and coldness of the limbs.

Her family history and surroundings were good.

The patient was a well-developed, well-nourished girl. The lips showed some cyanosis. There was no œdema. There was marked dyspnœa on exertion, great complaint of palpitation, pain over the precordium, and at times giddiness. The pulse was 92 per minute regular, of fair strength and moderate tension, the vessel wall normal. There was marked visible pulsation all over the precordium, especially marked in the left interspace internal to the mammary line, and in the second and third interspaces to the left of the sternum. The apex beat was in the fifth interspace internal to the mammary line. On auscultation at the apex there was a slight presystolic murmur, ending in a long blowing systolic murmur which was well conducted into the axilla and to the angle of the scapula. At the second left interspace, an inch to the left of the sternum, a loud double murmur was audible, the diastolic part of which was well conducted down the sternum. No murmur was audible at the

aortic cartilage. The respiratory system showed signs of œdema of the lungs. The urine was diminished in amount, but there was no albumin present. The other systems were normal.

For some time after admission to hospital the patient improved, but ultimately she developed an extensive thrombosis of the vessels of the right leg. The foot became gangrenous, and she died.

On *post-mortem* examination it was found that there was well-marked mitral stenosis. There was recent endocarditis affecting the tricuspid valve. The pulmonary valve cusps were healthy, but there was marked dilatation of the pulmonary artery, with incompetence of the valve. The pulmonary dilatation seemed to be secondary to the stenosis of the mitral orifice.

There are not many cases of pulmonary regurgitation to be found in the field of medical literature. Barié in 1891, and Gerhardt in 1892, gave detailed accounts of the literature of the subject, and since then a few additional cases have been published by Gibson, Bindeau, Kast and Rumpel, and Chaplin. The causes of pulmonary regurgitation, when not due to congenital disease, may be, as in the cases quoted, either an inflammatory affection of the valve cusps, or a secondary dilatation of the pulmonary artery from increased pressure. Of the inflammatory cases, in only about a quarter is a history of rheumatism obtainable. It is rare to meet an affection of the pulmonary valve alone; as a rule an additional lesion is present, usually aortic, and hence the differential diagnosis is greatly complicated. In the first of my cases there was an affection of the mitral valve, but the aortic cusps seemed quite healthy.

The second class of case, where the regurgitation is due to dilatation of the pulmonary artery from increased pressure, is rare, and one which has been but little recognised. Stokes has pointed out that in some instances there may be dilatation of the chambers of the heart without valvular lesion, and attended by dilatation of the pulmonary artery. Gouraud in his monograph expresses the same views as Stokes. Gibson, in a paper in the *Edinburgh Medical Journal* in 1880, advanced the view that increased pressure in the pulmonary artery might give rise to dilatation and consequent incompetence of the valve, and he has since been able to bring forward cases in support of the view.

Duckworth described a case of mitral and tricuspid stenosis, where, during the course of the disease, a pulmonary diastolic murmur developed, which was ascribed to pulmonary arterial reflux from dilatation of the pulmonary artery, entailing insufficiency of the valves.

Graham Steell has advocated the view that the "murmur of high pressure in the pulmonary artery" in mitral stenosis is by no means uncommon, but his views have not so far met with general acceptance. That the condition does occur there can, however, be no doubt, as, including my own, there are now several cases on record, verified by *post-mortem* examination, where, though the valve cusps were healthy, prolonged high pressure in the pulmonary artery had led to dilatation and insufficiency of the valves.

As an ætiological factor in the disease *age* seems to have little influence. It is true most of the recorded cases of pulmonary insufficiency are noted in young people, though Gerhardt had a patient of forty-eight suffering from the lesion. One must bear in mind, however, the gravity of the lesion. Once it is fully established, the outlook is a very serious one, hence most of the cases have been noted in young people, who may not long survive the rapidly occurring loss of compensation.

The *sex* of the patient varies. Gerhardt observed the condition most frequently in women, other observers have come to a contrary conclusion. The number of cases recorded is, however, too small to allow of any conclusion being drawn.

The *diagnosis* of pulmonary insufficiency is by no means simple. The very rarity of the condition is apt to mislead the observer, and drive him to the conclusion that the murmur is aortic in origin. In pulmonary insufficiency the diastolic murmur has its point of differential maximum intensity in the second interspace about 1 inch to the left of the sternum. It is conducted down the left border of the sternum. The murmur is rapidly lost on passing to the right of the mid-sternal line, but can be heard for a considerable distance on passing outwards and to the left from the point of differential maximum intensity. An aortic murmur loud enough to be heard out to the left axillary line must necessarily be audible at the aortic cartilage. The fact that the pulmonary diastolic murmur is not heard to the right of the sternum is probably due to the anatomical arrange-

ment of the pulmonary artery as shown by Ewart, and the relation of the right ventricle to the chest wall. Every physician must be familiar with the fact that the pulmonary second sound is heard over a very limited area of the chest wall. Thus in the case of a reduplication of the second sound at the pulmonary area, both the pulmonary and aortic sounds are audible; but on passing to the aortic area a single sound, the aortic, is, as a rule, alone audible. In pulmonary incompetence a diastolic murmur is audible at the second left intercostal space, if the aortic valves be competent a healthy second sound is heard at the aortic cartilage.

Some observers have remarked that the pulmonary diastolic murmur was affected by respiration, being louder during forced expiration, the increased loudness being due to increased pressure in the pulmonary artery during expiration. In neither of my cases was this phenomena noticeable, though in the first very special attention was paid to this point.

The audibility of the murmur in the back is of importance. Gerhardt advanced the view that in this lesion a double tone amounting almost to a murmur may be heard outside the border of the right scapula. In Case I. the murmur was very audible at the angle of the left scapula. I do not think, however, that any great diagnostic significance can be attached to this. I have carefully compared the diastolic pulmonary murmur with an aortic diastolic of similar intensity, and cannot find that the pulmonary is any better heard in this position or to the right of the vertebral column than an aortic diastolic murmur of a similar tone and intensity. Indeed, if we think for a moment of the anatomical arrangement of the parts, we would expect the aortic murmur to be the best heard of the two, as the aorta and left ventricle come into far more intimate relations with the dorsum than the pulmonary artery and right ventricle.

It has been stated by Friedreich that interrupted breathing is audible over the lung on deep respiration in these cases. The phenomena he considers due to the capillary pulse in the pulmonary artery. The observation and explanation are, I think, both to be received with some doubt. In neither of my cases could this be made out. The respiratory murmur was perfectly regular. When the phenomena does occur, I should think it is more likely to be due to a secondary respiratory

affection, and that it has no definite diagnostic significance for the lesion under discussion.

There is nothing characteristic in the pulse of pulmonary incompetence; it is rapid, weak and possibly irregular if compensation has broken down—the pulse of organic cardiac disease with want of compensation. This point is of importance in distinguishing the lesion from aortic disease.

The points then to be dwelt upon in the diagnosis of pulmonary incompetence are the location of the point of differential maximum intensity in the second interspace to the left of the sternum; the conduction of the murmur down the left border of the sternum; the area of audition of the murmur being wide downwards and to the left, very limited upwards or to the right; the absence of any characteristic alteration in the pulse, as is found in aortic incompetence. I do not think that much stress can be laid upon the alteration of the intensity of the murmur during deep respiration, nor upon the audibility of the murmur in the back. I was inclined, before I had seen a note of Gerhardt's observation, to think that the murmur was unusually audible over the scapula, but further observation and comparison convinced me that this was not the case. I do not think that any diagnostic significance can be laid upon irregularity in the respiratory murmur.

With regard to the question of prognosis, here as in other cardiac lesions we must take into account the general condition of the patient, his surroundings, temperament and station in life, the extent of the lesion, its effect on the heart, and the nutrition of the myocardium. Apart from these factors, pulmonary incompetence must be viewed as a very serious lesion. In aortic incompetence we have the strong left ventricle with its great power of establishing compensation, with, behind it, the mitral valve and the pulmonary circulation. In pulmonary incompetence there is only the comparatively weak right ventricle, with the comparatively less efficient tricuspid valve. Even in health the tricuspid valve is at times incompetent, a fact of which anyone can convince himself by riding a bike up a very steep hill, when the muscular effort will commonly produce a tricuspid systolic thrill. There is then in pulmonary incompetence but a comparatively slight barrier between the establishment of the lesion and loss of compensation with

backward pressure throughout the system. That this is so is borne out by the early age of the recorded cases, few having long survived the establishment of the lesion, and by the two cases which have come under my notice.

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## 2. SULPHONAL POISONING

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SULPHONAL is a drug so widely used, and generally taken with so imperfect a comprehension of its peculiar risks, that I have thought it well to publish the following fatal case of poisoning by it, which occurred some time ago, and to make it the text of some general remarks on the subject.

The patient was a man of thirty-nine years of age, who for nearly twenty years had exceeded in the matter of alcohol. He had, indeed, ruined his career by this failing, but his health had never been very seriously affected by it. He had, however, suffered from sleeplessness, for which he had taken various drugs at different times. About a month and a half before his death a medical man whom he consulted advised him to take thirty grains of sulphonal at night. This gave him excellent nights, and he seems to have taken the sulphonal every night from that time onward, so that one may calculate that he had taken at least 1200 grains, possibly more.

For about a week before his death it had been noticed that he staggered slightly in walking, and that his speech was thick and slurring, but, as he had been taking alcohol, no special importance was attached to these symptoms. On the 4th of the month his friends noticed that the urine was red in colour, and on the 5th he went to bed early in the afternoon, complaining of lassitude. On the 6th he remained in bed, and was noticed to be stupid and sleepy, but this, from his unfortunate habits, was so common an occurrence that not much was thought of it. On the 7th he was distinctly worse, and his attendants noticed that he had not passed water since the previous day. I was called to see him about 11 P.M. on that day.

I found him in a somnolent condition, when undisturbed sleeping quietly. It was quite easy to rouse him partially, and when roused he evidently understood what was said to him, and did what he was asked to do as far as he could. His motor power was seriously enfeebled, the grasp on the two sides equal but much impaired, and there was a corresponding weakness in all the muscles. He could not speak articulately. The pupils were of medium size, equal, and responded perfectly to light. The tongue was dry and red, with a slight fur in the centre. He had been constipated for some days, and a dose of Epsom salts had been given that afternoon, which he had promptly vomited. He had taken no food during that day, and seemed to find it impossible to swallow anything solid. His pulse was 90 per minute, fairly strong, and regular ; the heart sounds showed nothing special beyond a slight want of clearness in the first sound. The respirations numbered 24 in the minute ; there was some dulness with moist sounds at both bases posteriorly, and in the axillæ. The skin was cool and rather moist ; the temperature was 98°. The urinary bladder gave a dull note for about a hand's breadth above the pubes, and I passed a catheter easily and drew off about 25 ounces of urine, the first portion of which was of the colour of dark port wine, while the latter portion was more like a deep claret.

At this time I knew nothing of the history of the case, as I had merely been asked to see it for a brother practitioner who was ill, so that I ordered what seemed immediately necessary in the way of treatment, and went home, taking some of the urine with me to examine. When I found that neither blood nor albumin were present in it, it occurred to me that the case

might be one of sulphonal poisoning. Early next morning I went back to see him, and elicited from his friends the history I have narrated, which, not unnaturally, they had been reluctant to tell a stranger the night before. It had not occurred to them that there could be any relation between his symptoms and the sulphonal which he had been in the habit of taking, as the unfortunate man had several times assured them that it was a perfectly safe drug!

The patient was then in much the same state, but the pulse was distinctly weaker and somewhat irregular, and he died, rather suddenly, soon after I left him, remaining in the same semi-comatose state to the last.

The *post-mortem* examination was made twenty-four hours after death by Dr Robert Muir and myself, and the following are all the facts of importance we discovered.

The *liver* was somewhat chocolate-coloured, otherwise nothing special; gall-bladder full of bile.

The *kidneys* were somewhat congested, cortex normal in thickness, no evidence of cirrhotic change.

The *bladder* contained about half an ounce of red-coloured urine.

The *stomach* was in a state of granular catarrh and empty; no hæmorrhages in its walls.

The *spleen* was small, extremely congested, and almost diffluent; the Malpighian bodies were not very distinct.

The *suprarenal* bodies showed a cortex rather more yellow, and a medulla rather darker than usual.

The cavities of the *heart* were somewhat dilated, and the walls extremely fatty, especially that of the right ventricle; the valves were competent and healthy; the aorta normal but blood-stained; the blood everywhere fluid and chocolate-coloured.

The apices and anterior parts of the *lungs* showed no special change; the bases posteriorly seemed to be soaked in blood, were quite solid and of a dark purple colour, and sank in water at once; the bronchi were unaltered.

The *brain* was congested, markedly œdematous, the gray substance was pale and atrophied.

No other organ showed special change, and there were no changes that could well be ascribed to his abuse of alcohol, except, perhaps, the appearances in the brain; but I regret that the intestine and bone-marrow were not examined. Not

our will was to blame, but the fact that the examination was made under great difficulties in a private house.

Portions of the various organs were fixed, some in sublimate, some in alcohol, cut in paraffin, and examined microscopically by myself, in the Laboratory of the Royal College of Physicians.

The *liver* showed no evidence of cirrhosis. The cells were very granular, had usually two nuclei, and a fair number of the cells at the periphery of the lobules contained fat globules—a certain amount of fatty infiltration was present. The blood capillaries were sometimes very full of blood, sometimes empty. In the connective tissue of the portal spaces there were sometimes a good many leucocytes ; the walls of the branches of the portal vein were occasionally degenerated and hyaline. In the blood in the portal branches, but never in those of the hepatic artery or hepatic vein, were to be seen isolated liver-cells in fairly large numbers—far too many to have been swept in by the razor in the process of cutting. I am not prepared to explain this appearance : Dr Muir, to whom I referred the point, tells me that he has never seen this in the liver, but has sometimes seen a similar appearance in the lung in cases of pneumonia, and suggests that it is due to a partial disintegration of the tissue *post-mortem*, and that in the process of fixing and hardening, some of the cells are forced back into the vessels. Another possible, and to my mind more probable, explanation, is that the degeneration of the walls of the portal vein, which I have remarked on, may at some point have gone so far as to allow an opening into the vessel, through which the liver-cells may have passed into the blood-stream.

The glomeruli of the *kidneys* were congested, the epithelium of Bowman's capsule in some cases desquamated, while the epithelium of the convoluted tubules showed marked changes. The cells were almost all transformed into shrunken, granular lumps of protoplasm, without a trace of a nucleus, and were often cast off into the lumen of the tubules. The descending parts of Henle's loops were nearly normal, but the cells of the ascending limbs showed the same changes as in the convoluted tubules, save that they were not so advanced, and that one could here study the process of chromatolysis in the nuclei. The collecting tubules showed no marked change. There was no evidence of cirrhosis, the affection was purely parenchymatous.

In the *spleen* the Malpighian bodies were small and showed no sign of active leucocyte production. The spaces in the pulp were packed with red corpuscles, all in the state of "shadows," that is, without their hæmoglobin. There was very little pigment in the spleen.

The cells in the cortex of the *suprarenal capsules* showed extraordinary individual differences. In some of the columns the cells were small and shrivelled, in others they were enormously large, and consisted of a few threads of cytoplasm enclosing large vacuoles, while the nucleus was shrunken; some cells seemed to have lost their nuclei altogether. The cells in the medulla were small, granular, and shrunken. There seemed to be less pigment than usual in the gland.

That part of the *lung* which was solidified with blood was examined. The alveoli were found to be enormously distended with blood in the form of corpuscle-shadows and hæmoglobin-stained plasma, while some contained masses of leucocytes also. The alveolar capillaries were distended with blood, the alveolar epithelium seemed to be entirely lost.

The large pyramidal cells in the *brain* cortex were rather shrunken, but the organ showed no marked changes.

Unfortunately I had no opportunity of examining the *blood* during life, but films were made from the blood in the heart *post-mortem*, and these showed a certain amount of poikilocytosis in the red corpuscles. The deformed corpuscles were mainly microcytes. A few nucleated red corpuscles (normoblasts) were to be seen. The leucocytes were rather numerous, and curiously enough were mainly lymphocytes; the actual percentages in the differential count were:—

| | |
|---|---|
| Lymphocytes . . . . | 83 |
| Hyaline . . . . | 13 |
| Polymorphonuclear neutrophile . | 3 |
| Eosinophile, under . . . | 1 |

Dr Noël Paton kindly examined the *urine* with the spectroscope, and reported that both hæmatoporphyrin and urobilin were present.

In discussing this case I wish to take up—

1. The clinical history of chronic sulphonal poisoning.
2. The differences between this and acute sulphonal poisoning.

3. The pathology of the two conditions.
4. Their treatment.
5. The conditions under which sulphonal should and should not be used.

1. *Clinical history.*—One must carefully distinguish, as Friedländer (1) was the first to do, between the by-effects which may appear after patients have taken sulphonal, and which are due to a temporary effect of the drug, and those more serious symptoms which are the result of its cumulative action, and alone deserve the name of poisoning.

The former usually appear on the day after a medicinal dose of sulphonal, and include headache, tinnitus, muscular weakness, feeling of fatigue, continued sleepiness, giddiness, loss of co-ordination, and sometimes a scarlatiniform rash. The percentage of persons in whom one or other symptom occurs is not very large, and as a rule they pass off rapidly, with the occasional exception of the disturbance of co-ordination.

Chronic poisoning does not set in unless the patient has taken at least several medicinal doses, and usually not unless he, or she rather, for women are much more frequently affected than men, has been taking the drug for a month or so. Schulz's fatal case (11) was in a woman who had only had 15 grains of sulphonal sixteen times in a month—an allowance which many people exceed—but I can find no other fatal chronic case where so small an amount was taken.

As a general rule the first symptoms to appear are gastro-intestinal, anorexia, thirst, nausea, vomiting, and especially constipation, which may be followed by diarrhœa. There is sometimes cardiac weakness, there may be a skin-eruption; temperature does not seem to be specially affected, as both rise and fall have been observed. Soon after the gastric symptoms, as a rule, appear nervous symptoms, of which the most marked are ataxia of the extremities and a staggering gait, sometimes, though not so often, paralysis of the extremities or of facial muscles, and in very severe cases, localised or general convulsions. Mental apathy and depression, with anæsthesia of the skin, often come on along with the motor symptoms, and in the fatal cases pass on to coma before death. Drowsiness is not a very prominent symptom in most cases until the final stage is reached. Sometimes early, sometimes

later, appear changes in the urine, at first a diminution in its amount, then sometimes albuminuria, and especially that symptom which has come to be generally associated with sulphonal poisoning—hæmatoporphyrinuria—the excretion of deep-red urine, which owes its colour to the pigment hæmatoporphyrin. This urine is always intensely acid, and contains unchanged sulphonal.

Though it is evident that the hæmatoporphyrin must be derived from the hæmoglobin of the blood in some way, we know very little about the condition of the blood in these cases. Some observers have found a diminution in the number of red corpuscles, and a fall in the percentage of hæmoglobin, but the difference from the normal has seldom been very great, and many of the people in whom the poisoning occurred have been likely to be the subjects of a certain amount of anæmia. I shall have more to say of this in discussing the pathology of the condition.

The case which I have narrated was a fairly typical one, and terminated, as most of them do, by heart failure.

2. *The differences between chronic and acute sulphonal poisoning* are very considerable. The symptoms appear rapidly after a single large dose, taken by accident or for suicidal purposes as a rule. Sleep comes on, and sometimes a prolonged sleep lasting for days and followed for a time by giddiness, is the only evil effect. Sixty grains have produced a sleep lasting seventy-five hours, and in one case a woman who had taken 30 grains slept for forty hours, could not be awakened, and died at the end of that time!

In most cases which recover there are some after-effects —gastro-intestinal disturbance, muscular paralysis, ataxia, nephritis—which last for a longer or shorter time, and are not dependent on the amount which has been taken. It is curious, however, that hæmatoporphyrinuria only occurs, if it occurs at all, very exceptionally in the acute cases. With this may be noted the fact that in almost all the acute cases in which the blood has been examined no abnormality has been found, or if any change was noted it was simply a diminution in the percentage of hæmoglobin, which need not have been due to the sulphonal.

The prognosis in the acute cases is relatively much better than in the chronic ones; if treatment is begun early one can

almost depend on saving the patient, while a pronounced case of chronic poisoning can hardly recover.

To recapitulate briefly, the symptoms in the *chronic cases* are usually — first, gastro-intestinal, especially vomiting and constipation, then nervous, ataxia and depression, passing into a sleep-like coma, and with that albuminuria and hæmatoporphyrinuria. If the patient recovers these symptoms pass off, the hæmatoporphyrinuria being usually the first to disappear, and some nervous symptom generally remaining longest. In the *acute cases* the first and prominent symptom is sleep, and only when that has ceased do the other symptoms become evident. They usually pass off entirely after some time and leave the patient well.

3. *The pathology and pathological anatomy of the condition* depend on the facts that sulphonal is not readily soluble, that it may remain for a long time in the alimentary tract, that it or its products may also be retained in the blood and cause changes there, and that it is ultimately excreted by the kidneys. It will at once be seen that any weakness, functional or organic, along its line of march will favour the occurrence of chronic poisoning on the one hand, or add to the dangers of acute intoxication on the other. Thus constipation favours poisoning, and we find that almost all the fatal chronic cases have occurred in people who were habitually constipated. Any affection of the liver, of the blood or heart, and, at the other end of the chain, of the kidney, will assist in the accumulation of the sulphonal in the body and enable it to produce toxic effects. There is no doubt that accumulation takes place in perfectly normal people. Morro (5) has shown that after a few doses have been taken the amount excreted unchanged in the urine becomes larger every day, and when the drug is stopped, unchanged sulphonal is excreted in the urine for at least three days.

Let us follow the drug in its course through the body, and see what changes are produced in the different organs. The stomach and intestines are said sometimes to show superficial necrosis and Stokvis believed, from his experiments on rabbits, that hæmorrhages often occurred in the gastric and intestinal walls. These, however, are common in rabbits and not peculiar to sulphonal poisoning in them. In the liver, congestion has always been found, sometimes the cells are broken down, they

are always more or less fatty, but I have nowhere else found described the degeneration of the walls of the portal vein which I have noted. The heart has generally been found to be fatty, and from its weakness result the various signs of stasis which I have described in the lungs and spleen of my case, while Hoppe-Seyler and Ritter (2) refer the occasional broncho-pneumonia to the aspiration of food-particles into the lungs during the coma.

With regard to the changes in the kidney there is a striking unanimity of opinion. The chronic cases of Marthen (3) and Stern (9) showed exactly the same destruction of the secreting cells which I have described, the acute cases of Hoppe-Seyler and Ritter and others presented it, and it has been produced experimentally in animals; so that it seems to be an invariable phenomenon. It accounts, of course, for the oliguria and albuminuria, and for the tube-casts which have often been found in the urine of both chronic and acute cases.

The hæmatoporphyrinuria is much more difficult to explain, and has roused the interest of many observers. Hæmatoporphyrin, or iron-free hæmatin, is derived from hæmoglobin when it is acted on by acids in the absence of oxygen. Hæmochromogen is first formed, which then loses its iron and becomes converted into hæmatoporphyrin. It has been usual to assume that hæmatoporphyrinuria is causally associated with sulphonal poisoning and that it has to do with the frequent fatal issue, and to assume as a result of this that sulphonal is a blood-poison in the sense in which either chlorate of potash or anilin is a blood-poison. But it appears to have been overlooked that sulphonal can kill without the production of hæmatoporphyrinuria, and without causing any reduction in the number of red corpuscles or the percentage of hæmoglobin, for, as I have already said, hæmatoporphyrinuria does not appear in the acute cases, nor, in those acute cases in which it has been examined, has there been any change in the blood. In several of the chronic cases, it is true, anæmia has been found, but it has been always the kind of anæmia which is likely to occur in the course of a parenchymatous nephritis—a slight diminution of the red corpuscles and a rather greater diminution in the hæmoglobin, without any marked change in the leucocytes. The facts, besides the hæmatoporphyrinuria, which make one think of sulphonal as a blood-poison, are the

fluidity of the blood, and the ease with which the red corpuscles become transformed into shadows. The patients never looked specially anæmic, they were never cyanosed as in anilin poisoning, and it seems to me more than likely that the cause of death in both the acute and chronic cases is more probably to be sought in the kidney condition which is common to both, and which must certainly involve retention of products which should be excreted. It seems to me, in fact, that the cause of death is probably a uræmia, using the word in its widest sense, whose symptoms are masked by the effects of the drug. We must remember, too, that, as von Mering (4) has shown, it is impossible to produce hæmatoporphyrinuria at will, either in animals or man, even by giving large doses to animals for a long time, even though these animals were poorly nourished and acids were given along with the sulphonal, and the condition occurs in the most capricious way. Nakorai (7) has recently shown that Stokvis (10) was right in saying that hæmatoporphyrin was present constantly in the urine of patients suffering from lead poisoning, but the only other condition where hæmatoporphyrinuria is at all constant is in hæmorrhage from the intestine. Otherwise it is rare, though there seem to be small traces of hæmatoporphyrin in many normal urines. There seems a possibility that both in lead and sulphonal poisoning the hæmatoporphyrinuria may be due to intestinal hæmorrhage, but we know so little about the way in which sulphonal is absorbed, or even what are its decomposition products in the urine, that it is impossible to dogmatise on the matter. Kast's original view that it is excreted mainly as ethyl-sulphonic acid is not now widely held. Von Mering suggests that it may be excreted as amido-derivatives of ethyl-sulphonic acid. Certainly the urine is always strongly acid, and equally certainly, hæmatoporphyrinuria can be controlled by the free use of alkalies.

I am far from saying that sulphonal may not sometimes act as a blood-poison, and that the hæmatoporphyrinuria may not partly be due to that, but there must certainly be some other factor at work of which we as yet know nothing, and which determines that in one case a patient shall take sulphonal for months or years unharmed, while another dies in a month from the time the first dose is taken.

It is somewhat curious that in my case, though the kidney

was so seriously affected, no albumin was found in the urine by any of the ordinary tests. The explanation seems to me to be that there must have been complete suppression of urine-excretion for some time before death, probably for twenty-four or thirty-six hours, and that the urine which was drawn off by the catheter was the product of the kidneys before they became so seriously affected. It will be remembered that at the *post-mortem* examination only half an ounce of urine was found in the bladder, although it was about ten hours since the catheter had been passed, and that amount might easily have been left in the bladder after that operation. I suspect there had been complete anuria for some time before death.

I cannot explain the condition of the blood, especially the presence of so large a percentage of lymphocytes in the films, as I have made no special observations on the state of the blood found *post-mortem* in the heart. There certainly must have been some anæmia during life, or the few nucleated red corpuscles and poikilocytes would not have been present ; but equally certainly the anæmia cannot have approached in degree that in a pernicious anæmia, or even that in the acute anæmias, as, for example, that of septicæmia. It is, of course, possible that the sulphonal or its products may in some way alter the hæmoglobin of the corpuscles or alter the blood-plasma, without causing a very extensive breaking down of corpuscles ; but the mystery still remains unsolved, why it exerts its action as a blood-poison only in certain individuals, and only when it is allowed to act as a chronic and cumulative poison.

4. *The treatment of the two forms of poisoning* may be summed up as follows :—In the acute form, as the poison is absorbed but slowly, the stomach should be emptied if the patient is seen early enough, or the vomiting which is often present may be encouraged. Later, purgatives may be given to remove as much as possible of the drug from the intestine, and all possible means be tried to keep the kidneys acting freely. Neisser (8) saved his case, where 1500 grains had been taken in a single dose, by giving large enemata of warm water, frequently repeated, which produced free diuresis, and it would probably be well to try this again. Other treatment must be symptomatic.

In the chronic form, while it would be well to clear out the bowels and to promote diuresis, the best results have been

got by giving large doses of alkalies, as Müller (6) did, in such quantities as to render the urine alkaline if possible; probably both the bicarbonates and either the acetates or citrates should be given, but, as I said before, unless the chronic cases are very slight, the prognosis is very bad. Alkalies control the hæmatoporphyrinuria, and where their use has been stopped too early, the red coloration which had entirely left the urine has returned.

5. *Indications and contraindications.*—It is not worth while to speak of the indications, for I have made inquiries of several of the principal chemists in town and find that probably more sulphonal is used than all the other hypnotics put together, so that it evidently meets a felt want. It has the advantage of giving a very restful sleep; and as most people probably only use it now and again, so that plenty of time is allowed between doses for elimination, there is no great danger in its use in the majority of cases.

It should not be given in cases where there is great prostration, in cases where there is gastro-intestinal disturbance, especially if this takes the form of constipation, in cases of heart disease, in old people, or where there is a parenchymatous kidney lesion, acute or chronic. The maximum daily dose for a man should be 30 grains, for a woman 15 to 20 grains, and it should never be given continuously, but pauses of at least three or four days should be allowed from time to time to permit elimination of the accumulated drug. It should never be given in the solid form, both because it is less quickly absorbed, and acts less quickly in that way, and because it is more likely to cause gastro-intestinal disturbance. It is most soluble in hot alcohol, so that hot whisky toddy is probably the best medium in which to give it, the next best hot water.

The occurrence of any of the symptoms of chronic poisoning should be an indication to stop its use at once, and no patient who requires to take sulphonal for a long period should be allowed to pass out of observation. It is one of the disadvantages of the tabloid method of putting up drugs that patients can get such drugs as sulphonal without difficulty, and can continue their use without let or hindrance.

In cases where a hypnotic of the class to which sulphonal belongs is required, and where it is not desirable to use sulphonal, trional may with advantage be employed. Sulphonal

is di-ethyl-di-methyl-sulphone-methane, and trional is derived from it by the substitution of another ethyl radical for one of the methyls. It is much more soluble, and consequently acts in a smaller dose, more quickly, more certainly, and without leaving the unpleasant after-effects of sulphonal. Though it is not absolutely free from the risk of causing chronic poisoning, it is much less likely to do so than sulphonal, as it does not accumulate so much. Its only relative disadvantages are that it costs about twice as much as sulphonal, and that it has a more unpleasant taste. It is displacing sulphonal largely in Germany, to judge by the opinions quoted by von Mering, but shows little sign of doing so in this country.

I do not consider that sulphonal should be regarded as a dangerous drug, but I would plead for greater caution in its use, and especially for greater discrimination in the choice of the cases where it is to be employed. Its apparent capriciousness of action is its chief danger, and a drug which has a recorded death-roll of at least thirty cases, and probably many more unrecorded, is not one which should be recommended with a light heart to the first-comer.

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2. Hoppe-Seyler and Ritter, *Münch. med. Woch.*, 1897.
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4. Von Mering, *Therapeut. Monatsh.*, 1896.
5. Morro, *Deutsch. med. Woch.*, 1894.
6. Müller, *Wien. klin. Woch.*, 1894.
7. Nakorai, *Deut. Arch. f. klin. Med.*, 1897.
8. Neisser, *Deut. med. Woch.*, 1891.
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10. Stokvis, *Zeits. für klin. Med.*, vol. xxviii.
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## Meeting III.—December 21, 1898

SIR JOHN BATTY TUKE, *President, in the Chair.*

### I. EXHIBITION OF PATIENTS

1. *Professor Chiene* exhibited two cases of OSSEOUS DEFORMITY.

(*a*) C. S., age 11.

*Complaint.*—Weakness of back; rocking from side to side when walking.

*History.*—This condition has been present as long as patient can remember.

On examination, when patient stands erect the left shoulder is drawn up and left hip projects. There is marked lordosis. The line of the vertebral spines shows three distinct lateral curves. When she walks she has a slight limp, and swings the trunk and shoulders from side to side. The feet are somewhat flat. The length of the lower limbs is the same on both sides.

The power of abduction at the hip joints is limited, especially on the right side; all other movements at the hip joint are quite free. When the legs are rotated at the hip joint the head of the femur can be felt posteriorly in the buttock. This is more marked on the right side than on the left. The mammæ are unusually well developed for a girl of her age, and there is a marked depression at the lower end of the sternum.

Both shoulders droop forwards and inwards, and, on running the finger along the clavicles, a false joint can be felt in each clavicle about the junction of the middle and outer thirds of the bone. The inner fragment slightly overrides the outer. There is distinct mobility here, but no crepitus and no pain.

The friends state that when she was quite young she broke both her clavicles, one six weeks after the other.

*Diagnosis.*—Double congenital dislocation of hip joints, complicated with rotato-lateral curvature, probably rachitic; double ununited fractures of both clavicles.

(*b*) J. M'G., age 16, moulder.

Admitted Ward 13, Royal Infirmary, Edinburgh, 10th December 1898.

*Complaint.*—Pain in right knee on walking.

*History.*—Pain began in right knee about a year ago. He

can assign no cause for it. The pain was only felt when walking. It did not incapacitate him from work, but caused him to limp. At his work he kneels a great deal, but this does not cause the pain. In June 1898 he consulted a doctor, who said there was a swelling below patella, and applied a blister. After the blister healed the knee was painted with iodine and douched thrice daily with salt water. This treatment was continued for three weeks, and under it his condition improved. It soon, however, became as bad as ever, and on December 2, 1898, he again consulted the doctor, who sent him to the Royal Infirmary, Edinburgh.

Family history good.

Personal history good.

On examination, patient is a well-built, healthy-looking lad. When lying in bed he has no pain. When standing and bearing weight on the leg pain is complained of both on the inner and outer side of the right knee.

As he stands erect it is noted that the shoulders are on the same level ; the back is normal, there being no antero-posterior or lateral curvature. The chest is well formed.

The right side of the pelvis, or rather the right hip, appears more prominent than the left, but the muscles on the right side are not so large as those on the left.

When he walks he keeps the right leg somewhat rigid ; he does not take such big steps with the right leg as with the left, and brings the right heel down to the ground with a thump. On being asked to get out of bed he jumped down on the right leg, and this gave him no pain.

The right leg is everted, but he can stand without inconvenience with both feet together. There is no arching of the back when the right leg is extended.

Power of flexion is complete. On flexing the right hip, however, when the thigh is flexed until the right foot touches the left knee, it is found on continuing the flexion that rotation outwards of the thigh takes place.

Power of adduction is normal.

Power of abduction and circumduction much limited. Rotation outwards, as evidenced by the movements of the trochanter major, as free and through as big an arc as on the left side. Rotation inwards is limited. No pain is felt on carrying out these movements or on pressing on the trochanter.

The right gluteal fold is not so marked as the left; the muscles in this region are somewhat atrophied.

*Measurements.*—On measuring from the anterior superior iliac spine to the tip of the external malleolus there is found to be shortening to the extent of 1 inch on the right side; measurements from the tip of the trochanter major to the tip of the external malleolus are equal on both sides. Not only is there this upward displacement of the trochanter: it is $\frac{3}{4}$ inch nearer the middle line on the right side than on the left side.

There is no deformity of the pelvis.

*Diagnosis.*—Coxa vara, either due to late rickets, of which there was no other evidence, or possibly a slight injury to epiphysis.

2. *Mr F. M. Caird* exhibited—

(*a*) A patient, æt. 58, from whom he had removed 3 inches of RECTUM on account of carcinoma by the cava-sacral route. The growth was of nine months' duration, and occupied the greater part of the anterior and lateral aspect of the gut 3 inches above the anus. Its upper limit could be with difficulty reached. On 24th August a mesial exploratory laparotomy was performed and the upper limits of the growth defined, and the possibility of successful removal ascertained. The wound was then closed, and on the 6th September the usual incision dividing the sacro-sciatic ligaments was made, the coccyx was turned aside and the tumour removed. The divided ends of the gut were sutured after the method of Kocher, but sloughing occurred in the posterior aspect, and for some time the fæces passed through the wound. At present the patient is well and strong, defæcates naturally, and, save for a pinhole aperture, the wound is soundly healed.

(*b*) A patient, æt. 21, who presented symptoms characteristic of RECURRING APPENDICITIS and also to a slight extent of calculus in the right kidney.

On 14th July the appendix was removed. Its lumen was obliterated by granulation tissue and contained three soft fæcal concretions. On 4th October since, he still complained of dull lumbar pain, and there was constantly a trace of blood in the urine; the kidney was explored and an oxalate of lime calculus the size of a hazel nut was found in the upper posterior tissue of the kidney. He now enjoys good health.

3. *Dr Melville Dunlop* exhibited—

(*a*) A case of DOUBLE ATHETOSIS.

The child is a case of infantile cerebral paralysis. The condition is a congenital one, being probably due to cerebral atrophy or parencephaly.

The labour was normal, and there was no asphyxia present at birth to account for the condition. The symptoms manifested themselves shortly after birth.

The paralysis is of the diplegic type, but a certain amount of power has been regained, the child can walk with assistance and grasp objects feebly.

Tendon reflexes exaggerated and contractures are present.

Athetosis present in both feet and hands, and the movements are best seen on the child attempting to walk or seize anything. The walk is very characteristic of these cerebral conditions, being awkward and pawing with a tendency to cross-legged progression. The athetoid movements in the hands are complicated, slow, and deliberate, worse at times than others, and consist of extension followed by flexion of fingers, commencing at the little and gradually involving all the fingers. The movements continue during sleep. The muscles of the neck, face, and tongue are also involved.

Electrical reactions normal.

There have been no convulsions, the mental condition is only slightly impaired.

(*b*) A case of PSEUDO-HYPERTROPHIC PARALYSIS.

This case first came under observation sixteen months ago. The history was that he had been late in walking, easily got tired, and could never climb stairs without difficulty. The muscles of the calves, buttocks, and front of thighs hypertrophied, and the boy had the characteristic waddling gait, and climbed up from the recumbent position in the orthodox manner.

The condition has progressed very rapidly, and the boy can now walk only with great difficulty supported on either side. The muscles are hard, rigid, and respond only slightly to the foradic current. The tendon reflexes are abolished. The Pectoralis Major, Deltoid, Latissimus Dorsi, and Infra Spinatus are much wasted and atrophied, and the axillary fold is nearly abolished. When one attempts to lift the boy by the arm pits, he shows, in a well marked manner, the raising of the shoulders,

which takes place in these cases. The back is much arched when the boy attempts to walk, but the curvature disappears when he is seated. The boy walks with the feet in the position of pes equinus. The sensory functions are normal, and the mental condition fairly good. Peculiar choreic or athetoid movements are present in both feet and hands, to which he would like to direct the attention of the Society, and which he has not previously seen described. There are no fibrillar twitchings. A younger brother of the patient he (Dr Melville Dunlop) fears is developing the disease, the calf and thigh muscles being very hard and elastic and indiarubber like to the feel, and the gait is waddling.

4. *Dr Alex. James* exhibited—

(*a*) A case of SCLERODERMA. This patient, a mason, aged 34, had suffered for some months from symptoms like Raynaud's disease, so severe as to cause some loss of tissue at the points of the fingers and at the upper part of the tragus of each ear. The scleroderma seems to have commenced three months before. It was well marked over the hands and face, and, indeed, to a slight extent, over the whole body. In connection with the hands, it was sufficient to cause great difficulty in moving the fingers. On the face and neck it impaired his power of opening his mouth, and of protruding his tongue. The thyroid gland was atrophied, the spleen distinctly enlarged, and the liver slightly enlarged. There was no history of hæmaturia or albuminuria.

(*b*) A case of ANGIONEUROTIC ŒDEMA of one side of the tongue. The patient was a schoolgirl, aged 10 years. Her family history and surroundings at home were favourable. She had had measles and scarlet fever some years ago, and had otherwise been healthy. Her illness seems to have begun four years ago. After a severe chill, it was noticed that the right side of her tongue was swollen, rough, and sore, so that she could not swallow nor move the tongue properly.

With this attack there seems to have been salivation and enlargement of the submaxillary gland on the right side. This condition of the tongue lasted several weeks, and then subsided, leaving only a little enlargement of the right side of the organ. Since this first attack, however, she has had several similar

ones, coming on at intervals of five or six months, and preceded usually by chills.

Her present attack began about a week ago. She felt chilled and sick, and had a cough, and then the tongue began to swell. This swelling has gradually been getting worse, and there has been a flow of saliva with some blood from the swollen mucous surface. On examination she is a healthy looking girl, and complains of the continuous discharge from the mouth, and of difficulty in swallowing and speaking. Her pulse and temperature are normal. On opening the mouth, the tongue is seen to fill up the whole of its floor, but when protruded it can be seen that the enlargement mainly affects the anterior part of its right half. This part is fissured, ulcerated and bleeding at parts, and so swollen that a deep furrow occupies the place of the raphe. The consistence of this half is soft, and gives a feeling almost of fluctuation. The breath is foul, and the submaxillary glands enlarged and painful.

After admission, this condition gradually subsided, and in five weeks the child was discharged, all that was present being only a distinct enlargement of the right side of the tongue.

5. *Dr Graham Brown* exhibited, on behalf of Dr G. A. Gibson, two cases, one of BULBAR PARALYSIS and the other of PSEUDO-BULBAR PARALYSIS, presenting the marked and interesting symptoms of these two conditions.

6. *Dr J. O. Affleck* exhibited—

(*a*) Two patients, brother and sister, aged 13 and 9 respectively, exhibiting all the characters of FRIEDREICH'S ATAXIA. In the case of the boy the symptoms were typically displayed in the staggering gait, the choreiform movements of the arms, the curved spine, the semi-fatuous expression, and the peculiar speech. The knee jerks were entirely absent. The symptoms had been observed for years.

In the case of the girl the disease was not so far advanced, but the absence of knee jerks, the staggering, and the facial expression were well marked. Nystagmus was even more apparent in her than in her brother.

(*b*) A case of ADDISON'S DISEASE in a lad of 17, where

the symptoms, which were extremely well marked as regards the pigmentation of skin and mucous membranes, and the debility, had undergone considerable improvement after administration of supra-renal gland. The immediate effect of this treatment was some amount of fever and diarrhœa rendering it necessary to discontinue its use. But this was shortly followed by such an amount of improvement in strength and spirit as enabled the patient to return to his employment after having been laid aside for about nine months.

7. *Dr John Thomson* exhibited a boy, aged 9½ years, with HEMIATROPHY OF THE TONGUE (left half). He had always been tolerably healthy, and was said not to have had any illness except measles and whooping-cough. He has had no operation of any kind. It was not known when the tongue became effected. Its condition was first observed on his being brought to the Children's Hospital, when he was between 7 and 8, on account of dyspepsia. There had been no appreciable change in it since then.

The left half of the tongue was small and shrivelled. Its movements seemed defective, and it presented marked fibrillary twitchings.

The boy had a deformed palate, and was distinctly dull and backward in his school work, but there was no sign of any other local lesion.

8. *Dr George Elder* exhibited—

(*a*) A case of CONGENITAL HEMIATROPHY OF THE FACE, WITH MALFORMATION OF THE AURICLE. This boy, aged 7, has been in every way a healthy boy. The external ear was represented by a perpendicular ridge of cartilage, with several knobs and depressions. There was no auditory meatus, and the mastoid process was very imperfectly developed. Even when the other ear was closed he could hear when spoken to loudly. Associated with this was hemiatrophy of the whole of that side of the face, the lower part of the face suffering most, but the cranium also being deficient to the extent of about ¾ in. on the left half circumference of the head, as compared with the right. The malar bone, zygomatic arch, and lower jaw were all much smaller, and the palate was also badly developed on that side. Although in cases of malforma-

tion of the auricle, the lower jaw was sometimes less than on the normal side, it was apparently very rare for the hemiatrophy to be also marked in the upper part of the face and the cranium.

(*b*) A woman suffering from CHRONIC PROGRESSIVE CHOREA, WITH MENTAL ENFEEBLEMENT, BUT WITHOUT HEREDITARY TENDENCY. The patient, aged 54, first began to show symptoms of the disease after an injury leading to a colles' fracture of the arm. The movements began in the other arm, and gradually extended to the injured one, then to the face, legs and trunk. The movements were exactly like those of ordinary (Sydenham's) chorea, had got much worse during the last few months, and signs of mental enfeeblement had begun to manifest themselves. The movements ceased during sleep, decreased with voluntary action, but were exaggerated when patient was under observation. There was no loss of power, no affection of sensation, the knee jerks were exaggerated, the eyes were normal. The arms were most affected, but the face and legs, and to a less extent the trunk was also involved. Speech was somewhat affected, and occasionally there was a sudden peculiar inspiratory snort. The condition has lasted between four and five years.

9. *Dr Allan Jamieson* exhibited—

(*a*) URTICARIA PIGMENTOSA. A. S., æt. one year, Avonbridge. Admitted to Ward 38 in the Royal Infirmary in end of November 1898. A healthy enough child, with somewhat thin but soft skin, rather pale, and pouting expression. The eruption began on the face when he was two months old, as red patches or blotches, which gradually became more numerous, and spread over the body. It cannot be exactly ascertained if anything like a wheal first appeared. The mother states that the eruption did not itch much at the commencement, though it does now, to some degree. The eruption as now present consists of blotches varying in colour, from a pink or yellowish-pink to a deep brown, the older approximating most to the latter tint. Some, especially those on the upper part of the back have a faintly shagreen aspect. Most are flat, but a few are slightly elevated, and on pinching up the skin, it is found to be thickened. They are extremely numerous, many are roughly round, others are irregular in

shape. There are but few on the face, but many occur on the scalp, back of the neck, the chest—both in front and behind—and on the limbs. On the legs they are of a more purplish brown, on the arms redder. There are no evidences of scratching. The lymphatic glands in the neck are enlarged, and have a shotty feel. Permission could not be obtained to excise a portion of skin for examination, but the pathology of such cases is pretty well established, thanks mainly to the observations of Unna. There are found in the corium an enormous number of "mast cells," while the pigment in the basal cells of the rete is much increased. The condition is sufficiently rare, as only about sixty examples have been recorded. Treatment so far has had but little influence, but the discoloration usually fades, and the tendency to the production of fresh spots ceases, soon after puberty. The disease was first described by Nettleship in 1869, and there are apparently two distinct types, a nodular, which resembles xanthoma, and a flat and more pigmentary, of which this is an instance.

(*b*) PAPULO-SQUAMOUS ERUPTION, APPARENTLY SYPHILITIC, AFFECTING, AMONG OTHER SITUATIONS, THE PALMS. J. M., 46, railway cover maker, married, and has seven healthy children, the youngest aged 2 years. Admitted to Ward 37 on 14th November 1898. Denies any venereal infection, and no trace of a chancre could be discovered. The lymphatic glands were moderately enlarged. He states that a red, dry, itchy spot appeared on the front of the left wrist, in July last, and about a fortnight afterwards the body became covered with red spots. At the time this rash appeared, he took what he calls an attack of influenza, which showed itself by pain in the forehead and eyes, but there was no sore throat. A fortnight later the eruption spontaneously vanished. Sometime in the end of September a second eruption showed itself. It commenced in the same situation, but this time it attacked the palms, which became bright red, and their surface peeled. He was treated in the north with arsenic, and then with mercury, but not improving he came here. The eruption was found to affect the palms, arms to the elbow, margins of axillæ, and back of thighs. The skin of the palms was browish-red, glazed, and dry, so that he could not extend the fingers easily. The fingers indeed were flexed, somewhat swollen at the joints, and there were some fissures. The nails

were separated from their beds for fully one-third of their growth at the free extremity. Both elbows were covered with patches. These were in most instances round, from pea-size to a shilling, of a lean ham colour, this being more intense in the centre than at the edge. They were well-defined in contour, and bore whitish scales, but in no great amount. The skin underneath the scales was perfectly dry, thin, and shining. Many of the patches had a glistening aspect not unlike that of lichen planus, but there were no individual papules. There was much more pigmentary staining than occurs in ordinary psoriasis. The scalp was scurfy, and the hair thinning in patches. The treatment has consisted of poulticing the hands and arms with boric starch jelly, till the skin was fairly soft and clean. Then an ointment containing oxidised pyrogallic acid, salicylic acid, ichthyol and vaseline, was rubbed in daily. Internally small doses of perchloride and iodide were administered, with due precautions. Under this there has been a steady and continuous improvement. The patches have faded very materially, the palms are now soft, flexible, and free from fissures, while the general appearance as regards health is much better than on admission. The most interesting points here are the entire absence of any evidence as to how the poison entered the system, and the occurrence of the eruption on the elbows, a situation usually wholly avoided by the syphilitic papulo-squamous exanthem.

(*c*) PSOROSPERMOSIS FOLLICULARIS VEGETANS. Under this term, Darier in 1889 described a peculiar and rare disease, which, however, had been previously alluded to by Wilson and others. The disorder is a progressive and chronic one, and is characterised by the formation of small papules about the size of a pinhead, and not unlike those of lichen at first. These grow larger, assume a hyperæmic aspect, and develop a brownish or grey firmly adherent, conical crust. The lesions become confluent by the aggregation of new papules, and in this way extensive, somewhat elevated patches or areas are produced. These have a papillomatous appearance and are covered with thickish crusts. The disease is not unlike molluscum contagiosum, in a generalised form. It takes its origin at the pilo-sebaceous orifices, and in sections those bodies which have been thought by some to belong to the

sporozoa, by others to be mere degenerations of retained epithelium, have been found. The case submitted to you has considerable resemblance to Darier's Disease. The boy, aged 11 years, was admitted to his ward on the 24th of October, and the complaint had lasted for at least eight months. It began on the outer surface of the left arm, and has gradually spread to the other arm, the trunk, the lower limbs, and the face. When admitted there were to be seen small, flat, rounded papules, with a depression in their centres. Some of these became pustular, others became crowded together, so as to give rise to pinkish patches, raised above the surface, and with a glistening aspect, if not covered with thickish crusts, as were those on the arms and thighs. The disease presents differences from eczema, and is not molluscum, yet it has not entirely the characters of the complaint which bears Darier's name. Papules have been excised on two occasions, but sections were quite inconclusive, there was little if any morbid alteration perceptible. While he has been in the ward, he has at times been feverish at nights for a good many days in succession, possibly owing to the integumental irritation and pustulation. There was considerable itchiness, but this has disappeared. Under the continued inunction with salicylic vaseline, all the patches have flattened down, and there is now little more than pigmentary staining left to show where they were. The appearances now closely resemble those left by a fading lichen planus.

(*d*) RESULTS OF TREATMENT IN A CASE OF TYLOSIS, exhibited at the November meeting. This patient has been treated latterly with a strong salicylic and creasote plaster muslin, in place of the resorcin plaster muslin used at first. The latter caused the epidermis at first to exfoliate, but after a time ceased to effect this. As a consequence of the use of the salicylic and creasote muslin, the hands are now very nearly natural in appearance, and it is hoped that by the regular employment of a resorcin and salicylic soap, with friction, the overgrowth may now be kept within due bounds.

(*e*) CASE OF ELEPHANTIASIS. M. R., aged 43, native of north of Scotland, admitted to Ward 38, 16th December 1898. The patient has never been out of Scotland, and there is no history of any similar disease in her family. The complaint commenced in the left leg six years ago, insidiously, there

having been no attacks of inflammation or of erysipelas previous to its onset. Her hip joints are, however, anchylosed as a result of disease there for which she was treated thirty years since, in the Royal Infirmary, successfully by the late Professor Spence. The present ailment began by simple swelling, which slowly advanced upwards, till it approached the thigh, then the right leg took on the same condition. Till two years ago the skin of the left leg was smooth ; then a nodule formed on the calf, and now there are irregular prominences, half an inch in height, and somewhat depressed in the centre, each about the size of a bean. These are evidently made up of connective tissue overgrowth and dilated lymphatics. The skin from the knee almost to the ankle is covered with these prominences, and is further invested with dirty yellowish brown, ill smelling crusts. Much of the part seems affected with a species of chronic eczema. The skin of the right leg, on the contrary, is fairly smooth, at least at its upper part, but over the shin bears hard rough epidermic scales, in this resembling ichthyosis, with horny spinous accumulations, conical in shape, round the hairs. All the systems except the skin, are normal, and there is no enlargement of the liver or spleen. The blood has been examined after 8 p.m. by Dr Gray, but so far no filariæ have been found. As she has not been abroad, one could hardly expect to find them, but in view of the statement that they have been met with in Britain, a search was desirable, and it will be repeated. The curious element in the case is the occurrence without previous inflammation, or the presence of integumentary tubercular growths.

10. *Mr J. W. B. Hodsdon* exhibited a case of PLEXIFORM AND MULTIPLE NEUROMATA.

The patient, a female, aged 17, was recommended to Professor Annandale on 30th November 1898, and admitted into the Infirmary under his care.

The tumour, which was made up of a number of different lumps and stones, was first noticed about eight or nine years previous to admission, and had slowly increased in size. It extended high up into the axilla, and beyond the middle of the upper arm. The patient complained occasionally of shooting pains down the arm.

There were also a series of small nodules in the line of the

median nerve in the upper arm ; a well-defined almond-shaped tumour on the inner side of the ball of the thumb ; and a larger and elliptical well-defined tumour in palm of the left hand between the fourth and fifth metacarpal bone. The patient also showed well-marked patches of pigmentation over the trunk, face, and limbs.

In Professor Annandale's absence, Mr Hodsdon operated on December 7, 1898.

The tumour was found to extend from the bend of the elbow to beyond the coracoid process. It was unconnected with the skin, but adhered in parts to the periosteum.

The biceps muscle was displaced outwards by the growth, and the ulnar nerve and brachial artery backwards and inwards.

The median nerve passed through the centre of the tumour, which was removed in two portions.

The sheaths of the median and ulnar nerves were found to be enormously thickened, and to contain several nodular growths about the size of filbert nuts. These masses were removed. The wound healed *per primam*, and there had been no disturbance of nerve function, with the exception of two or three small bullæ on the inner side of the little finger, which were noticed the day after the operation.

## II. Exhibition of Specimens

1. *Mr David Wallace* exhibited—

(*a*) A large RENAL CALCULUS, which he removed from a patient, æt. 59 years. She had suffered pain in the right lumbar region, of a dull aching character, for eight years, but had manifested no other symptom, except hæmaturia on one occasion a month before operation.

(*b*) TWO KIDNEYS WITH CALCULI IN SITU from the same patient, she having died thirteen days after the operation. For a week after operation she gained strength and went on favourably, but then passed a quantity of blood per urethrum, and manifested severe gastric disturbance. She continued to pass a fair amount of urine—on day before death 23 ounces. In her supposed healthy kidney there was found *post-mortem* a suppurative calculous nephritis.

(*c*) THREE TUBERCULAR KIDNEYS after nephrectomy. Two of the patients recovered, and one of these is still living,

a year and four months after operation. The other died six months after operation. The third patient rallied well from the operation, but suppression of urine supervened on the third day, and lasted thirty-six hours, when secretion of urine again began, and continued until her death on the tenth day. At the *post-mortem* evidence of old general tubercular peritonitis was found, but there was no definite lesion discovered sufficient to account for death.

(*d*) A KIDNEY WITH MALIGNANT ADENOMA. The patient from whom this was removed suffered, from time to time for two years, from hæmaturia, but manifested no other symptom. The left kidney was palpable at the end of that time, and Mr Wallace performed nephrectomy. The patient made an excellent recovery, and there is no obvious return of the disease eleven months after operation.

(*e*) Portions of HYPERTROPHIED PROSTATE removed from three patients by supra-pubic cystotomy. In one case, a man, æt. 51 years, there was a well-defined middle lobe the size of a walnut which necessitated regular catheterisation. The residual urine was as much as 35 ounces—the patient being able to evacuate naturally only from 2 to 4 ounces of urine at a time. The bladder was atonic and flaccid with thin walls. He died nineteen days after operation. The urine was ample in quantity and of good specific gravity. It contained albumin. The pelves of the kidneys were dilated, but the kidney substance showed no recent inflammatory condition. In a second case, a man, æt. 58, the prostate was enlarged generally, but possessed a very prominent middle lobe of irregular shape which projected up and back into the bladder, producing a post-prostatic pouch of considerable depth. A calculus was present, and the patient suffered much pain and had to use the catheter very frequently both night and day. He made an uneventful recovery, and regained the power of expelling the urine. In the third case, a man, æt. 56, the urine was very septic and contained much pus. There was great frequency of micturition—20 ounces of residual urine. The patient had used a catheter twice daily for more than a year. Supra-pubic cystotomy was performed on 15th September 1898, and washing out employed for nearly three weeks. On the 7th November the second stage of the operation was carried out and the greater part of a circular prostatic ring round the urethral opening

removed. The prostate was very irregular but not ulcerated. It had contracted very little since the first operation. The patient made a good recovery.

(*f*) A FIBRO-ADENOMA FROM THE PROSTATE removed by supra-pubic cystotomy. The patient, æt. 58 years, had had bladder symptoms for three years, and used a catheter for almost two and a half years. Eighteen months before operation double vasectomy was had recourse to, but gave rise to no improvement in his condition. Catheterisation had to be continued, and he was troubled by frequency day and night. The prostate was very large, the right lobe in particular being apparently enormously hypertrophied. An incision was made through the mucous membrane over the mass in its long axis, and a tumour (fibro-endroma) the size of a small orange readily enucleated. He made an uninterrupted recovery, and regained the power of micturition.

(*g*) DERMOID CYST (Ranula) removed from under the tongue by an incision through the mucous membrane over the swelling. The cyst was readily enucleated.

(*h*) A LARGE GALL STONE with three smaller ones removed from the common bile duct. The patient, a woman, æt. 56, had jaundice at intervals for one year. There was an easily palpable swelling in the position of the gall bladder, which was, however, the left lobe of the liver—the gall bladder was much contracted and bound down by adhesions. She made a good recovery and has had no return of jaundice for eight months.

2. *Dr Logan Turner* exhibited A SKULL SHOWING AN UNUSUAL ARRANGEMENT OF THE FRONTAL SINUSES. Superficial examination suggested the existence of three sinuses, a mesial and two lateral. Closer observation, however, revealed the fact that the left sinus was almost completely divided by a vertical partition into two compartments, a smaller one occupying the mesial plane of the skull, and a larger one lying external to it. The partition was incomplete, there being a small aperture close to its lower end, so that both compartments opened into the left fronto-nasal duct. The septum between the right and left sinuses lay somewhat to the right of the middle line of the skull, and was vertical in position. He had not previously met with a similar subdivision. In the right frontal sinus, there was a small exostosis.

3. *Dr J. W. Dowden* exhibited—

(*a*) NAKED EYE AND MICROSCOPIC SPECIMENS OF PLEXIFORM, so-called neuroma, which was removed from the upper part of the right arm by Mr Hodsdon. The tumour was nodular and bound together by delicate connective tissue, and was about the size of a cocoa-nut; part was cleaned and dissected, showing the intricate branching and nodulation characteristic of the condition; the colour was light yellow, and resembled the appearance of a fatty tumour. Microscopic sections showed only fibrous tissue, there had not been sufficient time to obtain sections prepared in Müller's fluid.

(*b*) SPECIMEN OF HYDATIDS removed from the abdomen of of an Iceland woman, aged 39.

She had been complaining for the last eight years of steadily increasing abdominal swelling, and on admission to Professor Annandale's wards there was a distended condition of the whole abdomen from the xiphisternum to the pelvis and more marked on the left side, there was a very distinct thrill on palpation, and the note was dull all over.

Professor Annandale, to whom I am indebted for being able to show the specimens, operated on her, and evacuated the cyst. It contained many hundreds of smaller cysts, varying in size from a large cocoa-nut to a marble; there were many ruptured ones, and the fluid in the main cyst was turbid, yellow (about a gallon of it was squeezed out), showing under the microscope cholestrine and fatty globules. The edges of the cyst wall were stitched to the abdominal wall, and free drainage provided, the patient progressing favourably.

4. *Mr C. W. Cathcart* exhibited—

(*a*) A case of MULTIPLE CEREBRAL ABSCESS OF EMBOLIC ORIGIN.

J. C., æt. 36, was treated in Ward 5 for empyema. A portion of rib was removed, and the cavity drained. In a few weeks he was sent to the convalescent hospital, making a satisfactory recovery, but after about a week's residence he was sent back in a comatose condition, and died next day. We learned that up to the second day before he was sent back he had been going on well. On the first day that he was taken ill he vomited once or twice in the morning, but seemed otherwise well for the rest of the day. Next day he vomited several times,

and complained of severe frontal headache. In the evening his temperature was 103.3 ; respiration, 34 ; pulse, 108, full and bounding. No explanation could be found of these symptoms. Next morning he could not be roused, and had passed urine in bed. He was therefore sent at once in an ambulance to Ward 5 again. We found him absolutely unconscious, showing symptoms which could not be satisfactorily explained on the theory of meningitis, cerebral hæmorrhage, or embolism. There was a raised temperature, full pulse of 84, and slight tendency towards Cheyne Stokes respiration ; occasional twitchings of the hands and fingers, but no indication of paralysis, and no spasm of muscles of back of neck or elsewhere. The diagnosis made was that of cerebral œdema, with a slight amount of meningitis. At the *post-mortem* examination the white and grey matter of the cerebrum was studded over with spots of red softening varying from the size of a pin's head to that of a hazel nut. There was also a slight amount of basal meningitis. Dr Shennan found twisted clumps of strepto-cocci in films prepared from these points of red softening. They were evidently early stages of abscess formation. In culture he found strepto-cocci in short chains, also staphylo-coccus pyogenes albus and a bacillus like that of the coli communis.

There were no signs of recrudescence of the lung trouble, nor of endo-carditis or breaking down clot in the heart. The abdominal viscera were not examined. It was remarkable that while the spots of red softening were scattered throughout every part of the cerebrum, the cerebellum, pons, and medulla had completely escaped.

(*b*) ABDOMINAL ANEURISM, causing difficulties in diagnosis.

The patient, D. M., a seaman, aged 46, had been treated in Aberdeen Royal Infirmary some months before he came to Edinburgh. His case had been obscure, and was discussed by Dr Ashly Mackintosh in the *Scottish Medical and Surgical Journal* for March 1898. The chief difficulty while he was in Aberdeen had been caused by the occurrence of symptoms pointing to the kidney, followed by the appearance of a tumour in the iliac fossa, which was at first solid, but afterwards pulsated. When he came to the Royal Infirmary, Edinburgh, he had a large pulsating swelling in the left iliac fossa, and another in the lumbar region of the same side, but it was difficult to make out if they were continuous. Opinions were

divided as to whether there were two aneurisms, an abdominal and an iliac ; a single abdominal aneurism which had extended downwards, or a single iliac aneurism which had extended upwards. He died of exhaustion, and at the *post-mortem* examination it was found that the condition was due to a single abdominal aneurism. This had affected only a small area of the posterior wall of the aorta at the level of the cœliac axis. Apparently the sac had burrowed at first downwards, and to the left, lifting up the kidney ; afterwards it had burst downwards, and the mass of blood thus escaping had formed the iliac tumour, which was at first solid. This mass had afterwards softened, and the interior becoming continuous with the original aneurism, had thus secondarily pulsated. The primary and secondary sacs were quite distinct, with only a narrow aperture between them. Apparently a third stage in the development of the aneurism had been a diffuse extension backwards. This had been the direction in which the swelling had increased latterly. There was no sac in this part of the aneurism corresponding to the rapid increase which had been observed clinically.

5. *Mr F. M. Caird* exhibited preparations to illustrate an unsuccessful result of EXCISION AND SUTURE OF GUT FOR CARCINOMA, in which gangrene of portion of the intestine had occurred.

The patient, a woman æt. 58, had symptoms for eight months. There was a tumour the size of a goose's egg in the left iliac fossa. It was rather fixed and tender.

On 27th September it was removed, and the dilated upper end was sutured to the narrowed lower end. The mucous membrane was not quite so vascular as usual. It was necessary to remove a good deal of the mesocolon and the adjacent parietal peritoneum which were adherent and involved, but the greatest care was taken to preserve intact the collateral circulation of the bowel. A few appendices epiploicae were stitched over the line of suture to make all secure, and a drainage tube was inserted. The patient progressed most favourably, and on the 1st October passed flatus and a good motion. After this, however, spontaneous action of the bowels ceased, and enemata were required. Pus appeared to a slight extent, she suffered from retention and some nausea, although she had no evidence of peritonitis. On the 4th October the wound was freely

opened up and found to contain fæces. She died on the following morning. The sectio, obtained with difficulty and somewhat hurried, showed localised collections of pus in the pelvis and left iliac fossa, the latter with fæcal matter. The line of suture was perfect, but there was complete gangrene of the gut from a point (at which it had given way) from about 1½ inch above the line of union to an unascertained extent below. The gangrene had probably taken place gradually after the fourth day, and may have been due to some kink in the vessels or thrombosis of the weak vascular supply consequent upon the peristalsis.

6. *Dr William Elder* exhibited—

(*a*) INTESTINE AND HEART from a case of dysentery with ulcerative endocarditis.

Patient, a man aged 32, admitted to Leith Hospital on September 13th and died on the 16th. On admission he looked very ill, was in a semi-collapsed condition, and was suffering from dysenteric diarrhœa. This diarrhœa continued more or less till his death in spite of astringents both by the mouth and by the rectum.

He had also a well-marked systolic murmur heard all over the precordial region, but loudest over the sternum, where there was also a diastolic murmur. His liver was enlarged considerably, and he was very breathless and required to be propped up in bed. He died on the fourth day after admission, but it was noticed that the character of the heart murmurs changed towards the end of his life. A diagnosis was made of dysentery and ulcerative endocarditis. The temperature on admission was 100. It fell to 97 next day, but rose again to over 100 immediately before death.

The intestine at the *post-mortem* showed small ulcers with congestion and small punctate hæmorrhages in the mucous membrane of the descending colon and sigmoid flexure.

The heart was hypertrophied, especially on the left side, and there was ulceration of previously calcareous valves.

Patient had had rheumatic fever when sixteen years of age, and had had breathlessness and palpitation on exertion occasionally since.

(*b*) BRAIN WITH HÆMORRHAGE INTO THE OCCIPITAL LOBE, from a man, aged 38, who was admitted to Leith Hos-

pital on September 10th, 1898, in an insensible condition. He had been going to his work when he suddenly fell down insensible.

On admission he had paralysis on the right side of his body. Left pupil dilated, right contracted, and neither reacted to light. Later, however, it was found that both sides were much the same, and movement of his right side was produced by sharp pinching. A diagnosis was made of a cerebral hæmorrhage, but not in the usual position. It was impossible to test his eyesight.

Patient became more and more comatose, and died the day after admission.

*Post-mortem* revealed a large flattened out blood clot immediately beneath the dura on the left side spread over the middle two-thirds of the hemisphere and extending down to the base and the sylvian fissure. It was seen to have come from near the tip and under side of the occipital lobe. The hæmorrhage had first taken place into the cortex and subcortical tissues of the occipital lobe, and had then ruptured through the cortex and spread over the hemisphere down to the base of the brain. The hæmorrhage came from a branch of the posterior cerebral artery.

(*c*) BRAIN FROM A CASE OF WORD BLINDNESS. A man, aged over 60, consulted him on the 17th November, nearly a month before he died. When first seen he was dull and stupid-looking and had general enfeeblement. He had no localised paralysis. He remained in much the same condition till the 29th November, when it was found that he had slight motor paresis on the right side of the body, and at the same time it was found that he had word-blindness and partial letter-blindness.

On the 2nd December he had slight motor paresis of right side, no anæsthesia, no hemianopsia, could hear and understand quite well. He could repeat words perfectly; he could speak voluntarily, using correct words; knew the uses of objects and could name objects. He could not read more than a few letters, and very few small words. In attempting to read he spelled, but did not use the proper letters nor the proper number of letters. For instance, when asked to read—Like the English the Spanish language has four moods—he said: c i m i n m i o c i m i m n i n m i n i m c i m o. He had

thus a form of letter intoxication. He could only write a few letters, voluntarily and to copy, but could write a little better to dictation. Died on 11th December.

The *post-mortem* examination revealed a considerable amount of atheroma of the cerebral vessels, more marked on the left side than the right. The vessels at the base were practically converted into calcareous tubes. The vessels all over the left hemisphere were thickened and calcareous and looked in some places like pieces of cord. There was a patch at the posterior end of the sylvian fissures which was softened. This patch included exactly the angular gyrus and extended from the interparietal fissure above to the posterior end of the first temporo-spheroidal convolution, and from the posterior end of the supra-marginal convolution in groups to the first occipital convolution behind. It was circular on the cortex and about 1½ inches in diameter. On cutting into this patch it was seen to pass in a wedge shape till it reached the posterior horn of the lateral ventricle, which it just reached. The cause of the softening was found to be a thrombus in the terminal branch of the sylvian artery. There was no affection of Broca's convolution, nor of the motor areas or auditory centre, and there was no other affection of the brain. The word blindness was therefore due to softening of the angular gyrus.

## III. Exhibition of Instruments

1. *Mr C. W. Cathcart* exhibited his MICROTOME, on which certain improvements had recently been effected. A holder had been made for the knife, and was adapted for an ordinary razor. The holder was steadied by overlapping side-pieces, and thus could not slip off laterally. Instead of the glass plates on which the plane iron used to rest, and which were constantly becoming loose, polished brass plates were now used, and on these the razor-holder worked. They were fixed with screws and were quite secure. A rachet had been adapted to play against the milled head and indicate by its sound the amount of turn given to the raising screw. The size of the milled head had been increased and the number of teeth forming the milling been counted. Each tooth as it passed the rachet now indicated that the screw had been raised $\frac{1}{8000}$ of an

inch. By these means the microtome did its work more simply and more exactly than before. Mr Fraser, the maker, had carried out the suggestions for these improvements with his usual skill, without adding much to the expense of the instrument. Formerly it cost 21s., including provision for cutting specimens embedded in paraffin; now the cost of the whole would be about 27s.

2. *Mr F. M. Caird* exhibited a METAL NEEDLE AND SUTURE BOX which he had devised for the purpose of sterilising silk sutures by boiling. The sutures are kept apart, do not entangle, and are lying seriatim ready for use.

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## Meeting IV.—January 18th, 1899

Dr P. A. YOUNG, *Vice-President, in the Chair*

### I. ELECTION OF MEMBER

Andrew Messer, M.B., C.M., Lenington, Scotswood-on-Tyne, was elected an Ordinary Member.

### II. EXHIBITION OF PATIENTS

1. *Mr D. M. Greig, Dundee*, exhibited a man, aged 34 years, the subject of congenital and symmetrical perforation of both parietal bones. The case had previously been described by Mr Greig in Vol. XXVI. *Journ. Anatomy and Physiology*, but the man had not previously been shown at any society, and Mr Greig was of opinion that this is the only case which has been recognised during life. The patient suffered no inconvenience from his deformity, and had served for seven years as a driver in the Royal Artillery, and later on for three years as a private in the Seaforth Highlanders, with which regiment he saw active service in India. The deformity consisted in an osseous defect nearly 4 cm. by 3 cm. at the posterior superior angle of each parietal bone. Through these unossified areas the pulsation of the brain could be felt, and severe pressure caused pain, dizziness in the head, and flashes of light in both eyes. They corresponded with the anterior part of each superior occipital convolution.

Mr Greig was of opinion that the defect was due to a maldevelopment about the parietal foramina.

2. *Messrs F. M. Caird and W. Guy* exhibited a patient, an ex-private of Hussars, who had suffered from untreated syphilis five years previously, and in whom most extensive destruction of the bones and soft parts of the face, nose, and mouth had resulted. The hard and soft palate, the nasal and alveolar processes of the superior maxilla, the soft parts and cartilage of the nose and a great part of the upper lip had disappeared. The vomer, ethmoid, and turbinated bones were also involved. There was one common naso-pharyngeal cavity, and the finger could be passed to the cribriform plate of the ethmoid, and backwards to the sphenoidal cells. The patient improved under treatment, and the active progress of the disease being arrested, Mr Caird requested Mr Guy to undertake the prosthesis of the case.

An artificial nose and upper lip were made in vulcanite, and retained in place by means of a spectacle-frame. The glasses were removed from the frame because the patient's sight was good, and because of the difficulty he would experience in wiping them in wet weather. An artificial palate was also made. Into this, and emerging from it in the middle line so as to just impinge upon the back of the artificial upper lip, was vulcanized a strip of springy gold, which could be adjusted to slide in a box catch fixed at the centre of the back of the upper lip. The nose piece being in place the gold strip was bent in such a way, that the palate was fixed in true and just position, when the extremity of the strip was lodged in the catch. An artificial moustache was fastened to the lip, the appliance was coloured in oils. The result from an æsthetic point of view may be judged from the photographs. The speech which had been absolutely unintelligible, could now be made out, but the improvement in this respect, though distinct, was hardly so rapid as is usual in similar cases.

## III. Exhibition of Specimens

1. *Dr Harvey Littlejohn* exhibited—

(*a*) Two specimens of hypertrophy of the heart.

In the first, the heart weighed 29 ounces. The pericardium was universally adherent, and the aortic and mitral valves were

APPEARANCE OF PATIENT BEFORE PROSTHESIS.

APPEARANCE OF PATIENT AFTER PROSTHESIS.

TO ILLUSTRATE PAPER BY DR CAIRD AND DR GUY.

thickened. It was obtained from a man 21 years of age, who had had two attacks of rheumatic fever at 7 and 14 years of age respectively. He had, since the last attack, enjoyed good health, and was able to bicycle and take other forms of exercise. Death occurred suddenly in bed while apparently in the best of health. In the second case, a man, 35 years of age and 5 feet 10 inches in height, dropped down dead in the street. He left the army six years ago in good health. During the last 18 months he was twice under treatment in the Royal Infirmary for cardiac symptoms. He had recently been discharged from the hospital, and was to enter on the duties of a situation he had obtained the next day. The pericardial sac was distended with ¾ of a pint of clear fluid, and the heart itself was enormously hypertrophied, weighing 47 ounces. The aortic orifice was dilated. The liver and kidneys were cirrhotic.

(*b*) A SPECIMEN OF SPONTANEOUS RUPTURE OF THE LEFT VENTRICLE, from a woman 63 years of age, who on getting out of bed suddenly complained of feeling ill, with shortness of breath and precordial pain, and was found dead, lying upon the floor, 20 hours afterwards. She was very stout, and the heart was covered with fat on its external surface. On laying open the left ventricle, the anterior ventricular wall was found to be ruptured about 1 inch above the apex, the rupture running transversely for 2½ inches and dividing the columnæ carneæ and muscular substance to a depth varying from ⅛ to ¼ of an inch. The muscular tissue showed advanced fatty degeneration and infiltration on microscopic examination.

(*c*) AN EFFUSION OF BLOOD UPON THE SURFACE OF THE DURA MATER, resulting from traumatic rupture of the middle meningeal artery, and illustrating the advantages of Jores' method of preservation for museum and educational preparations.

## IV. Original Communications

## THE LOCALISATION AND SYMPTOMS OF DISEASE OF THE CEREBELLUM CONSIDERED IN RELATION TO ITS ANATOMICAL CONNECTIONS.

By ALEXANDER BRUCE, M.A., M.D., F.R.C.P.Ed., Assistant-Physician, Royal Infirmary, Edinburgh.

IN bringing this communication before the Medico-Chirurgical Society to-night, my main object is to draw attention to the

importance of certain nerve tracts, which have been proved by the recent investigations of neurologists to bring the cerebellum into intimate relation with the spinal cord and higher parts of the central nervous axis.

I do not propose to approach the subject from the point of view of the anatomist, but rather with the intention of impressing upon the clinician that a knowledge of their position and relations, when considered in connection with the results of experiment on the cerebellum and its peduncles, may afford valuable data for the explanation of some of the symptoms of cerebellar disease, and may assist him in the difficult task of its localisation.

The symptoms of cerebellar disease may for our purpose to-night be divided into two classes: first, those which are without special significance beyond the fact that they indicate the existence of an intra-cranial lesion, and which have no further localising value; and, secondly, those which are regarded as being more definitely characteristic of affections of the cerebellum. Among the former may be placed headache, optic neuritis, hebetude, and vomiting; among the latter the disturbances of equilibrium and co-ordination indicated by the peculiar reeling gait, the ataxia, the severe vertigo, and the deviations and oscillations of the eyeballs.

One important and puzzling feature of cerebellar disease is that these characteristic symptoms, as is well known, are not invariably present in all cerebellar lesions, for some such lesions may run a course which is more or less latent, or may be indicated by symptoms of so vague a nature as, in the present state of our knowledge, to be practically useless for localising purposes; and on the other hand similar symptoms may result from lesions which do not affect the cerebellum directly. One of the first steps towards the explanation of these peculiar variations in the phenomena of cerebellar disease was the recognition of the fact that these differences in the symptoms are not due to differences in the nature of the lesion (occurring as they do equally in tumour, hæmorrhage, softening, and sclerosis), but depend, as Nothnagel was the first to show, on differences in its situation. This author states in his *Topische Diagnostik der Gehirnkrankheiten* that "the conclusion to be drawn from all these observations is that the situation of the lesion is the factor that determines the

presence or absence of symptoms; only when the lesion is limited to one hemisphere can it run the course without symptoms" (p. 47). "There are certainly many cases which show that lesions in the hemispheres may also produce symptoms . . . ., but the analysis of these cases shows, in our opinion, without doubt, that the lesion in such cases involved adjacent parts in one way or other." It may be concluded, therefore, that the loss of the substance of one hemisphere does not of necessity produce any symptoms, or at least any symptoms as yet known to us. "Lesions actually limited to one hemisphere cannot be diagnosed" (p. 48). "Disturbances of co-ordination only incur when the lesion indirectly or directly involves the vermis—that is, the middle lobe of the cerebellum" (p. 50).

"Another question (p. 51) is whether disturbances of co-ordination must invariably appear when the vermis is involved. In my earlier publications I answered this in the affirmative on the strength of the observations then at my disposal."

"The later observations show undoubtedly that tumours may be situated in the vermis without causing any symptom of cerebellar ataxia, and therefore lesions of the vermis need not be invariably associated with disturbances of co-ordination.

"In this connection I may mention that these exceptions are always cases of tumour—that is, of slowly-growing lesions, and that in all cases a greater or less part of the vermis was left intact."

Nothnagel is frequently credited with the unqualified statement that it is only tumours in the middle lobe that cause disturbances of equilibrium, and that tumours of the lateral lobes always run a latent course. From what has just been quoted, however, it will be seen that this is not a correct interpretation of his opinion.

I shall now endeavour to explain from the anatomical standpoint why it is that disturbances of equilibrium occur specially in connection with lesions of the middle lobe, and why in certain instances these disturbances may also be present in lesions of the lateral lobe. The anatomical data have been obtained partly by the collation of observations of various investigators on the human fœtus, partly by the study of degenerations following experimental and pathological lesions of the brain and cord.

The cortex of the middle or vermiform lobe of the cerebel-

lum forms the central termination for at least six important tracts derived from the spinal cord, two arising from the cord directly and the remainder from the nuclei of the postero-median and postero-external columns, namely, the clavate and cuneate nuclei (two arising from these nuclei on the same side,

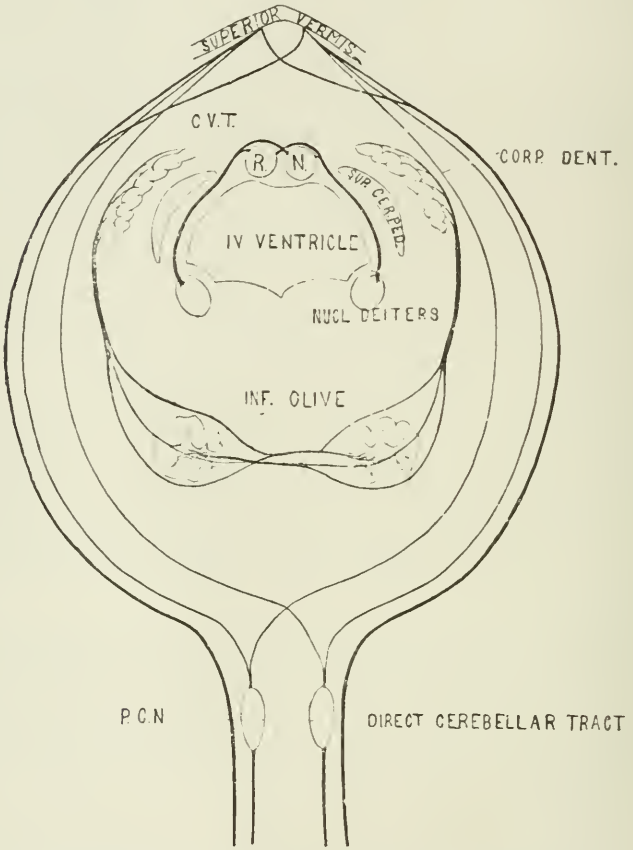


Fig. 1.—The constituents of the inferior peduncle. P. C. N., Each oval area represents the combined nuclei of the two divisions of the posterior columns, and gives a double tract represented by a single line to the restiform body of its own side and another to the opposite side. These represent the interrupted paths from the posterior column. R. N., roof nuclei. C. V. T., cerebro-vestibular tract from roof nuclei to nucleus of Deiters.

and two from those of the opposite side, each represented by a single line in Fig. 1). The uninterrupted fibres consist of the direct cerebellar tract and the ascending antero-lateral tract of Gowers. All these tracts, except the last mentioned, are con-

tained in the restiform body or inferior peduncle of the cerebellum.

The tract of Gowers passes further up the pons and hooks round the fifth nerve before bending into the vermis, as has been shown by Loewenthal, Mott, and Tooth in animals and by myself in man. The majority of the fibres terminate in the upper part of the middle lobe, some on the same side and some after crossing the middle line.

While it must be admitted that the functions of these tracts are still somewhat doubtful, it is certain that they are not concerned in the conduction of impulses which give rise to impressions of touch, temperature, or pain, since they may be destroyed without interference with these forms of sensibility, and it must be presumed that some, if not all, of them, are concerned in the conduction of muscular sense impressions. It is important to note that none of these tracts pass to the lateral lobes, nor does any other tract from the cord, as far as is known, enter the lateral hemispheres or become connected with them, except indirectly, and even then to a comparatively slight extent. These tracts are all undoubtedly afferent to the cerebellum.

Another tract which was at one time regarded as a purely afferent sensory tract, and was therefore termed by Edinger "the direct sensory cerebellar tract," connects the nucleus of Deiters (one of the end nuclei of the vestibular nerve, lying at the lateral angle of the fourth ventricle), with the roof nuclei of the cerebellum.

Until quite recently the relations of Deiters' nucleus remained very obscure. Its most characteristic feature, and one that has impressed itself on most observers, is the large size of its cells, which in appearance are practically identical with the motor cells of the spinal cord. So close is this resemblance that, from the first time my attention was directed to these cells, I felt convinced that they must belong to a motor nucleus, although at that time it was not possible to trace their connections. Subsequent research has, however, determined to a very great extent the destination of the fibres derived from this nucleus. Although our knowledge of its relations is still far from being complete, enough has been learned to indicate its great importance. Monakow, in 1883, showed that the cells of this nucleus became atrophied after hemisection of the cord in the cervical region. The significance of this fact was,

perhaps, not at first fully apparent, but the discovery of the existence and nature of the neuron showed that its cells must give origin to fibres which passed down the cord, and which had been divided in the above-mentioned operation. This view had been actually advanced by Deiters himself long before this.

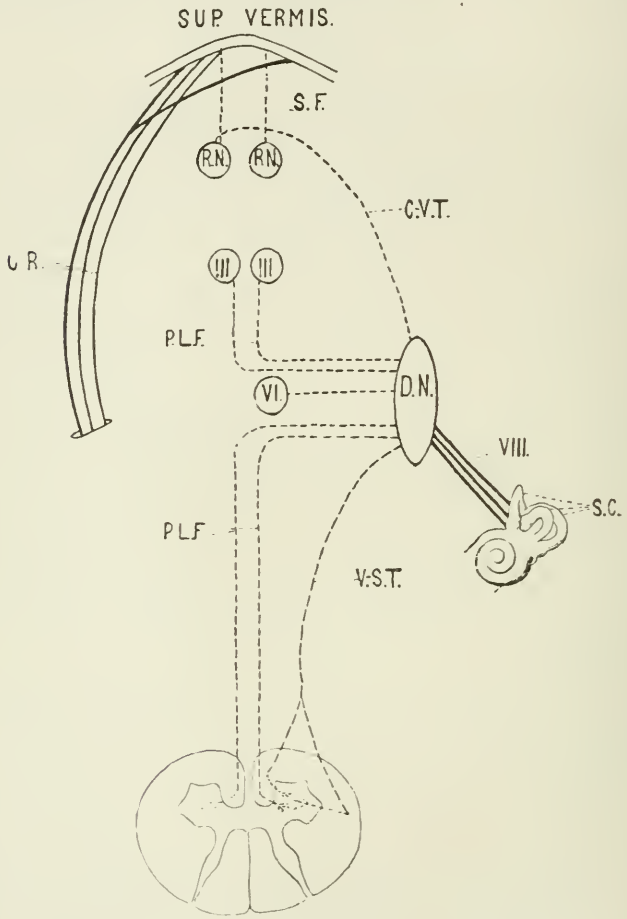


Fig. 2.—Scheme of the connections of the nucleus of Deiters.

In 1888-89-92 I described two tracts arising from this nucleus; of these one passed in a downward direction, and could be followed as far as the outer side of the inferior olive; but, owing to the method of investigation then employed, it was not possible to trace it further.[1] Held subsequently observed that

[1] *Proceedings of the Royal Society of Edinburgh*, xvii., 1888-1889, p. 26.

it was continued downwards into the antero-lateral columns of the cord, and more recent investigations have confirmed this view. The other tract passed inwards under the floor of the fourth ventricle, and, after giving a certain proportion of its fibres to the sixth nucleus, it crossed the middle line and apparently passed upwards in the posterior longitudinal fasciculus as high as the third nucleus. More recently our knowledge of the connection of these two tracts has been further extended by the study of sections stained by Marchi's method after recent injury to the nucleus of Deiters.[1] It is now known that the first tract—which may be called the vestibulo-spinal tract—passes downwards and enters the same side of the cord, where it divides into two parts, one division taking up its position at the side of the anterior median fissure and the other in the lateral column. Risien Russell has traced some of these fibres as far as the lower end of the thoracic region of the cord. They terminate in the anterior cornu of the cord on their own side at various levels.

The second set of fibres, that which passes inwards under the floor of the fourth ventricle, divides into several groups. One of these terminates in the sixth nucleus of the same side; the remaining groups enter the posterior longitudinal fasciculi (P. L. F.) on both sides, some turning upwards and some downwards. The two groups which bend upwards terminate in relation to the two third nuclei; those which have a descending direction pass down the anterior columns of the cord, where they occupy a position at the margin of the anterior median fissure. They appear to terminate by entering the anterior cornua of the cord.

It will be seen that, by means of these various tracts, the nucleus of Deiters is placed in a position to influence the nuclei of the ocular nerves and the motor cells of the cord throughout the greater part of its extent.

The nucleus of Deiters has another very important connection that can be traced backwards from it along the side of the fourth ventricle, then across the mesial plane of the cerebellum to terminate in a nucleus which is called, from its position in relation to the fourth ventricle, the roof nucleus. This tract was formerly regarded as the continuation into the cerebellum of the auditory nerve, and even of the lower cranial

[1] Ferrier and Turner, Risien Russell, and André Thomas.

sensory nerves, and, as already said, was termed the "direct sensory cerebellar tract" by Edinger, but Ferrier and Turner, and more recently Risien Russell and André Thomas, have shown that the tract has its origin in the roof nucleus, and is thus not an afferent but an efferent tract, arising in the middle lobe of the cerebellum, and terminating in Deiters's nucleus.

When the middle lobe of the cerebellum is further examined (Figs. 2 and 3), it can be seen that its cortex is connected by a series of sagittal fibres with the roof nuclei. This important link completes a chain of afferent, internuncial, and efferent fibres between certain sensory tracts in the cord, the middle lobe of the cerebellum, the nucleus of Deiters, the various oculo-motor nuclei, and certain motor centres in the cord. The integrity of this chain appears to be essential to

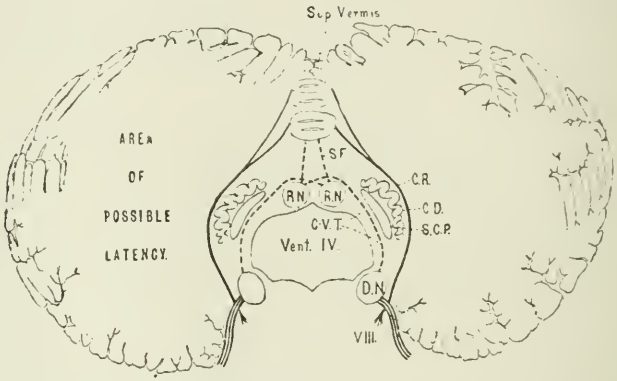


Fig. 3.—Diagrammatic transverse section of cerebellum to show connections of middle lobe: C. D., nucleus dentatus; S. C. P., superior cerebellum peduncle; C. R., inferior peduncle.

the perfect maintenance of equilibrium. If we follow the course of the fibres in this system (as indicated in Fig. 2), we see that an impulse would travel upwards through the restiform body (C. R.) and Gower's tract (not drawn) to the cortex of the middle lobe of the cerebellum (superior vermis). It would then pass by means of the sagittal fibres (S. F.) to the roof nuclei, (R. N.). Starting again from one of them (the connection with the other roof nucleus being ignored for the sake of simplicity), it would pass by the cerebello-vestibular tract to the nucleus of Deiters. Here there might be a reinforcement by other impulses travelling from the semicircular canals (S. C.) by way of the vestibular nerve (VIII). At Deiters's nucleus fibres to the nuclei of the sixth and third,

and perhaps the fourth nerve, as well as to the anterior cornua of the cord, mainly on the same side, would be stimulated, and in this way there would be put into operation a very complex mechanism which is well adapted to affect the equilibrium of the body. We can thus understand how irregular or defective stimulation of Deiter's nucleus may lead to lateral oscillations of the eyeballs, and to irregular or impaired movements of the same side of the body, resulting in vertigo and disturbances of equilibrium.

It will be seen from what has just been said that the cerebellar termini of the constituents of this system are in the cortex and roof nuclei of the middle lobe; no such connection of the spinal cord or its nuclei with the lateral lobes of the cerebellum can be made out. These lobes seem to receive afferent fibres from the nuclei in the pons, which, in their turn, are in relation with the cortex of the frontal and temporal lobes of the cerebrum.

If we now consider the system of the superior cerebellar peduncle, which is also undoubtedly concerned in the maintenance of equilibrium, we see that this peduncle arises mainly from the nucleus dentatus, and terminates above in the red nucleus and optic thalamus of the opposite side. The nucleus dentatus is situated at the junction of the middle and lateral lobes, and is connected extensively with both of these portions of the cerebellum, and also through the inferior peduncles, with the inferior olives of the medulla (see Fig. 1).

It will be observed from Fig. 3 that this nucleus is in such a position as to be readily affected by lesions involving either the middle or the lateral lobes. It will be seen, for instance, that a section dividing the middle from the lateral lobes will cut through the nucleus dentatus, thus severing the origin of the superior peduncle, and may at the same time cut across both the afferent and efferent tracts of the middle lobe—namely, the inferior peduncle and the cerebello-vestibular tract. From these facts we can readily understand why it is that removal of one-half the cerebellum or of one lateral lobe causes practically the same symptoms as removal of one-half of the middle lobe, since in both cases the afferent and efferent fibres of one side of the middle lobe are divided, and the superior cerebellar peduncle arising from the nucleus dentatus is also involved to a greater or less extent. We are also in a position

to understand why a section in the mesial plane of the middle lobe may cause few symptoms of disturbances of equilibration, and why these disturbances may be readily recovered from. Such a section will destroy the afferent and efferent fibres from both sides to an equal extent, so that the resulting interference with equilibration will be merely a symmetrical weakening of that function on both sides. This can be compensated for to a great extent by that portion of the mechanism for equilibration which has remained unaffected by the lesion. For the same reason a tumour of the vermis occupying a symmetrical position with reference to the middle line, or at least which destroys, symmetrically, afferent and efferent fibres of the vermis, would be marked by few or no symptoms, especially if its growth were so slow as to allow of compensation. The pronounced symptoms in the case of unilateral lesions are due undoubtedly to the fact that the interference with the mechanism is limited to one side, and the results produced are therefore asymmetrical. If one cerebello-vestibular tract or one nucleus of Deiters is alone affected, the usual stimuli from the latter to the nuclei of the ocular nerves and to the anterior cornua of the corresponding side of the spinal cord will be wanting ; thus we can understand (1) the yielding of the limbs of the same side and the tendency to fall to that side, and (2) the oscillatory movements of the eyeballs.

These symptoms may, however, be absent after lesions of the lateral lobe external to these tracts, which would leave these systems entirely, or practically entirely, intact. Turner,[1] in reviewing Luciani's work, states that "in our experiments rotation round the longitudinal axis certainly occurs more constantly when the peduncles are divided than when the lateral lobes are removed. But this is not an absolute rule, and one of the most marked instances of rotation round the vertical axis which we observed was an animal in which only the cortex of the lateral lobe had been cauterised and under conditions likely to excite irritation of neighbouring parts." This statement may be regarded as being so far in harmony with the view just enunciated.

Thomas[2] expresses himself even more unequivocally: "Lesions involving a considerable extent of the cortex, but not extending deeply and not implicating the central grey

[1] *Brain*, 1884, p. 7.

[2] *Le Cervelet*, p. 317.

nuclei, produce disturbances which are of relatively short duration and of moderate intensity. Lesions limited to the cortex of one hemisphere, and not extending deeply, may be free from any disturbance."

It is well known that section of one superior or inferior cerebellar peduncle produces rotatory movements and other motor disturbances, and also that similar disturbances follow removal of one half of the vermis or of one lateral lobe. This second fact may be readily understood when the arrangement of the parts is considered. An examination of Fig. 3 shows that removal of one half the vermis or of one lateral lobe involves a section of the intracerebellar portion of these peduncles. It is also stated that section of the middle peduncles produces somewhat similar symptoms, and since the cerebellar terminations of this peduncle are limited to the lateral lobe, it will naturally be asked why removal of the lateral lobe, which must involve an intracerebellar section of the fibres of this peduncle, should not in like manner produce similar symptoms. In answer to this, I would suggest that many of the results ascribed to section of the middle peduncle are really due to involvement of the upper or lower peduncle, or of the cerebello-vestibular tract, or of all of these, during the operation employed for division of the middle peduncle, and that the results of a section involving only the middle peduncle have yet to be determined.

If the views here adduced be well founded, we may expect disturbances of equilibrium to be produced by symmetrical lesions situated within an area bounded by the intracerebellar path of the two inferior peduncles, of the two superior peduncles, and the dentate nuclei in which the latter arise. This area contains the middle lobe (superior and inferior vermis, the roof nuclei, and the sagittal fibres connecting the latter with the cortex), and the cerebello-vestibular tracts from the roof nuclei to the nucleus of Deiters (Fig. 3). Lesions within this area may produce no such disturbances, provided they are symmetrically situated with reference to the mesial plane, and especially if their growth is so slow that compensation is established *pari passu* with the disturbances they may tend to cause.

On the other hand, lesions situated in the lateral lobes may produce no disturbance of equilibrium, provided they are situated entirely external to the intracerebellar paths of the upper and

lower peduncles, and of the nucleus dentatus (*area of possible latency*, Fig. 3). If, however, these structures are interfered with, either by pressure or by direct involvement, then the characteristic symptoms of cerebellar disease will be produced, and will depend in their character and amount on the nature and extent of this interference.

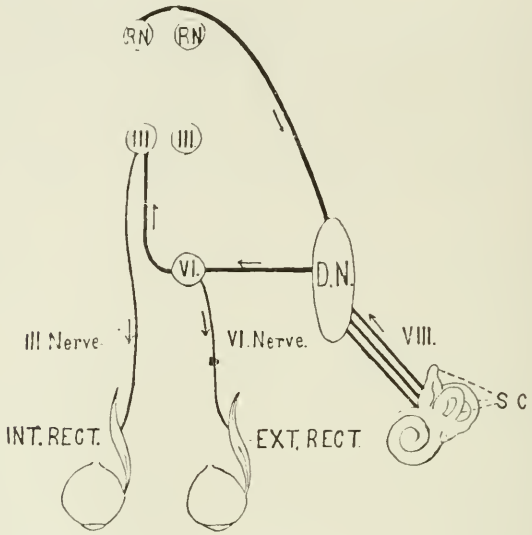


Fig. 4.—Relation of Deiter's nucleus to the mechanism for conjugate deviation.

If the cerebello-vestibular tract, or Deiters' nucleus, be injured, then the usual stimuli will not pass either to the anterior cornua of the cord or to the sixth (fourth) or third nuclei. Hence may result the weakness of the same side, the tendency to fall to that side, the impairment of the conjugate deviation to that side, the tendency of both eyes to be directed to the opposite side, and the lateral nystagmus which occurs, especially when the eyes are directed towards the same side. A glance at Fig. 2 will show how the spinal symptoms may be produced, since the nucleus of Deiters will not act through the vestibulo-spinal tract and posterior longitudinal fasciculi upon the anterior cornua, and more especially upon that of the same side. The effect on the position and movements of the eyes will be easily understood from Fig. 4, if it be remembered that the sixth nucleus is the nucleus for the conjugate deviation of the eye to the same side, supplying, as it does, not only the

external rectus, but also, through the opposite third nucleus and nerve, the internal rectus of the opposite side. A stimulus of Deiters' nucleus, acting on the sixth nucleus, will turn the eyes to the same side; absence of this stimulus will allow the other sixth nucleus to pull the eyes towards the opposite side, and will thus weaken the voluntary attempt to look to the same side. On the other hand, lesions which merely irritate but do not destroy the nucleus of Deiters may cause rigidity or spasms of the same side of the body, or such an excess of movement as may cause the body to fall to the other side, and by acting on the sixth nucleus may turn the eyes to the same side.

Luciani, Ballance, and Thomas have also drawn attention to the possible effect which the superior peduncle may exert through the red nucleus and the optic thalamus on the ocular nuclei and the opposite motor nucleus, and it is not my intention to question this influence in producing the symptoms of the cerebellar disease. At the same time, the importance of the nucleus of Deiters as a co-ordinating centre seems too evident to be ignored.

It is by no means claimed that the above described mechanism is as yet so fully understood as to be capable of explaining all the symptoms of cerebellar disease, but it, at least, affords a working hypothesis on which the most characteristic and frequent among them may be interpreted.

## 2. ON A CASE OF CEREBRAL ABSCESS ILLUSTRATING CERTAIN FORMS OF APHASIA

By Alexander Miles, M.D., F.R.C.S., Edinburgh, Surgeon to Leith Hospital, and Theodore Shennan, M.D., F.R.C.S., Edinburgh, Pathologist to the Hospital.

The following case appears to us to be worthy of being put on record as an illustration of one of the ways in which the cerebral speech mechanism may be interfered with by a collection of pus in the temporo-sphenoidal lobe. The abscess was secondary to middle ear suppuration, and for a time ran the usual clinical course of such cases. It was evacuated, and the patient's urgent symptoms disappeared. On the following day, however, it was observed that he showed symptoms of paraphasia and "word intoxication." For the next two months he

continued to exhibit peculiar forms of interference with speech, notably an inability to apply the correct names to objects, a tendency to employ the verbal instead of the nounal form in identifying objects, and frequent use of unintelligible language, although there was no word-deafness.

The patient was a man, 22 years of age, and was occupied as a labourer. He was admitted to Leith Hospital on May 13th, 1896, complaining of pain and swelling in the region of the left ear. There was nothing in his family or personal history to throw any light on the condition from which he suffered. Three months before admission—in February 1896—his left ear began to "run," but this did not trouble him much, except from the accompanying deafness. Sometimes there was a good deal of discharge, then for a time there would be none. Four days before admission he caught a "severe cold," and on the next day the left ear became sore and painful with swelling all round it. The pain was so great for the next two days as to prevent sleep.

On admission this painful swelling was noted particularly in front of the ear and over the mastoid. Soon after admission relief was obtained by the escape of a large amount of discharge. There was no vomiting, giddiness, or loss of sight. Hearing was markedly diminished on the left side. On the 14th the left side of the head was slightly more prominent than the right. The swelling was greatest in front of the tragus, but it was not now tender. There was no facial paralysis. On the 15th the patient had a bad night from pain on the left side of the head. The ears were examined: the left was found to contain much yellow fluid pus, and the membrana tympani was obscured by granulations; the right ear contained wax. The patient heard the ticking of a watch on the left side only on contact, and better on mastoid contact. It was heard by the right ear at a distance of 18 inches, and on median contact was better heard by this ear. The disease therefore was probably still only in connection with the middle ear and mastoid cells. On the 16th there was pain on both sides over the forehead, but no giddiness or staggering. The temperature reached 102·5° F. There was no rigor and very little discharge from the ear. On the 17th there was less pain and only on the left side of the forehead. On the 18th the pain and discharge were less, and there was tenderness over

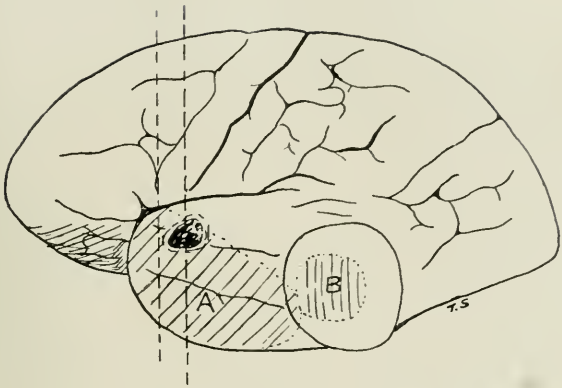


FIG. 1. OUTER SURFACE OF LEFT HEMISPHERE.

A. Extent of primary abscess.
B. Pedicle of hernia cerebri, dotted line; outline of hernia indicated by the firm line.


FIG. 2. POSTERIOR SURFACE OF A TRANSVERSE SECTION FROM THE POSITION SHOWN IN FIG. 1, SHOWING THE EXTENT OF PRIMARY AND SECONDARY ABSCESSES.

1. Primary abscess.
2. Secondary abscess.
Is. Island of Reil.
C. Claustrum.
L. N. Lenticular nucleus.

TO ILLUSTRATE PAPER BY MR MILES AND DR SHENNAN.

the mastoid. On the 22nd an operation was performed. The mastoid antrum was opened and the middle ear was scraped out. The fluid injected by the mastoid opening passed out by the external auditory meatus, but not very freely. There was not much pus, and but little smell. The patient had a good deal of pain in the afternoon. At 3.30 P.M. half a grain of morphia was administered subcutaneously. Improvement continued till the 28th, when the patient became quite insensible, very noisy and restless, and had to be tied down. At 10.30 P.M. on that date the patient was trephined over the posterior part of the temporo-sphenoidal lobe. With an aspirator needle pushed forwards and slightly downwards, pus was at once struck, and it escaped freely. The wound was further opened up, drained, and dressed with iodoform gauze. The pus was in the temporo-sphenoidal lobe, as well as in the meninges. This rapid abscess formation was the first evidence of intracranial mischief. Hitherto no aphasia had been noted.

On the 29th the patient, though dull and stupid, was quite conscious. He was irritable and anxious to get up. There were evidences of paraphasia and word-intoxication. He called a pipe a "knife" and matches "knife." There is a certain association of these objects with the idea of filling and lighting a pipe. It is interesting to note that there has been abscess formation in the temporal lobe without causing aphasia or any special localising symptoms. The only symptom noted is the great pain on the left side, particularly over the forehead, but that, along with the rise of temperature, occurred a fortnight before operation. On the 30th the patient was still restless and anxious to get up. Marked "paraphasia" was present. When asked what day it was he answered, "10 o'clock." He asked to be allowed to get up for "two weeks," meaning two hours. When shown a watch he called it a "clock," but he could not read the time correctly. He knew that his answers were incorrect and indicated by gestures his annoyance at this. In the evening he complained of pain at the back of the "right nose" and asked for the "night office," meaning the house surgeon. On the 31st he was still confused but looked better. He used wrong words for everything but wrote well to dictation and copied writing fairly well, therefore no word-deafness or visual aphasia existed. He understood what he was writing and copying; there was no paragraphia. During examination

he became irritable and refused to stay in bed. On June 1st he determined to get up on to the "drawer," meaning the sofa. On the 3rd he was still anxious to get up, took food better, and had a good appetite. He could tell the time on a watch but could not name a key or a pair of scissors. On the 4th at about 4 A.M. he somewhat rapidly became dull and stupid and complained of great pain in the centre of the forehead and over the left eye; he kept groaning, did not know where he was, and could not understand what was said to him. He answered questions with great confusion of words so as to be quite unintelligible. At 3 P.M. he was still dull and drowsy. Q.—"Have you any pain?" A.—"Yes." Q.—"Where?" A.—"I'm all right." Q.—"Where is the pain?" His answer to this question was confused gibberish spoken very deliberately. On dressing the wound it was found that the pus was not draining freely. A probe passed inwards, downwards, and forwards gave exit to a quantity of thick, stinking pus, apparently extra-dural, over the roof of the tympanum. After the dressing the patient spoke freely but did not appreciate or use correct words. Q.—"What day is this?" A.—"Six o'clock." Q.—"What time do your friends come?" A.—"Twenty past six." Before these questions were put, he heard the resident surgeon ask the nurse if this was visiting night and when his friends came, therefore to some extent these answers were suggested. The condition of the patient had become worse on June 3rd, so that while, as formerly, he could not name a pair of scissors he could not now even express the abstract idea of cutting as being their use, and the report of the following day (June 4th) bears this out. With the paraphasia there was now for a short time some evidence of word-deafness, so that he talked gibberish and did not appreciate his mistakes, indicating an affection of the auditory ("word-hearing") centre, probably simply by pressure, as the exit of pus relieved the condition for a time. This is not negatived by the correct answer "Yes" to the question as to pain, as this is one of the very common words for the appreciation of which the right hemisphere may be educated.

On the 5th at 11 A.M. the wound was dressed. Drainage was still imperfect. In the afternoon the patient complained of great pain. At 8.15 P.M. he was much worse; he presented a pale-gray colour and was drowsy. The pulse was 60.

The wound was explored with a director and a good deal of pus escaped both from the cerebral and the extra-dural collections. Slight left facial paralysis was observed. After dressing the pulse was irregular, varying from 60 to 72 per minute. Here there is evidence of general compression, looking to the relief obtained by the evacuation of pus, and affection of the facial nerve as it passed in relation to the middle ear. From 10.30 P.M. to midnight the patient was restless and noisy, calling out loudly, sometimes singing out his words. There was almost constant trembling of the whole of the left side, his right hand and arm were cold, and both his eyes were slightly open while he was asleep. At a quarter past twelve o'clock morphia was given hypodermically. On the 6th at 4 A.M. there was great pain which could not be localised. The patient talked incoherently. At 5.30 A.M. he was quieter again. The wound was dressed at midnight. The patient uttered no complaint of pain but said that he felt better. He was sensible. The pulse, which was irregular, was from 60 to 80. On the 7th, at 4 A.M., the patient was awake. Q.—"are you better?" A.—"No." Q.—"Will you take some milk?" A.—"Give me some 'Diest,' the stuff I had before" (meaning beef juice). Slight tremor of the left hand, arm, and leg was observed. The pulse was 90 and regular. At 6 A.M. the patient, who was awake, looked bright and understood everything which was said to him. He spoke freely and sensibly, but could not give some things their proper names—*e.g.* wanting paper he asked for "bread." The pulse was 84 and regular. The patient had slept well all night and was not drowsy or stupid when awake. At 7 P.M. he could read words —*e.g.* "Edinburgh"— correctly. "Society" he pronounced *sokiety* and "Scottish" *skottidge*. When asked to name figures in a picture he seemed to understand what was wanted, but gave absurd names to things, mixing up syllables so as to make words of no meaning. There was no word or figure-blindness, but there was paraphasia in answering. On the 8th the improvement continued. He slept well, felt well, and spoke to the other patients. He read a little for his own amusement, said he felt "fine," and wanted to get up. When shown a pencil he called it a "kencil." A safety-pin he called a "nail-cut," a knife a "knife," a key a "knife," a bunch of keys "kniffs" (knives), and a watch a "handle." He could tell time

on a watch correctly. There was no word-deafness, as shown by his ability to read for his own amusement. On the 9th there was still some paraphasia. On the 12th a hernia cerebri of the size of a small hazel nut was noticed. On the 13th the patient complained of some pain in his eyes.

Notes of the case from this date onwards to June 25th give evidence of continued improvement. The patient still misnamed things, but in some instances after a time he would recall the proper name as if there were slow cerebration. He was able to get up and to walk about. On the 26th he played a game of dominoes. When asked to play he said that he did not know what it was, but when once he began he seemed to pick up the game very readily, and he played correctly. This note is interesting, as indicating that the centres necessary for making the moves—visual—and the tracts connecting these with the motor, and the motor centres themselves were not involved in the disease, being placed higher than the parts destroyed by the abscess. It is also interesting that he read and understood the newspapers, as it indicates that not only the centres immediately concerned in this act but also the special word-hearing centre was not involved, as a patient cannot read with understanding when this centre is destroyed. On the 28th he went out and continued improvement was noticed on the next day (June 29th). When shown a pencil he said: "Always forget the name of that," and when asked what it was for he explained by signs and said that it was "for writing." A safety-pin was shown to him and he said, "I don't mind the name *yet*." He said it was for "catching anything," making a sign with his sleeve. He could not name a box of matches, but said it was for "lighting pipe." He could read the words "paraffin matches" on the box. When shown a match after reading the name on the box he could not name it, and when asked if it was a match, he said, "I don't think so." The last note is curious, as it would mean the existence of some word-deafness or auditory aphasia, but, as Dr William Elder, who saw the patient, suggested, as he had paraphasia the answer "I don't think so" may not have been what he intended to say. Though he could read the words "paraffin matches" he evidently did not understand what they signified. The former answers indicate no word-deafness at all. It is quite possible that had he been shown the words "pencil" and "safety-pin"

he would not have been able to connect them with the objects which they denote.

So far the case indicates clinically a lesion affecting the conducting fibres from the auditory centre to Broca's convolution. At times the history is difficult to understand, as some symptoms—the gibberish, &c.—seem to indicate word-deafness, and shortly after others would indicate that this did not exist. From this date onwards the patient grew worse. On July 4th the hernia cerebri was much smaller, nearly flush with the surface, and the discharge was slight; elastic pressure was applied pretty firmly. In the evening he began to complain of pain in the head, and he vomited greenish-yellow fluid. He had a peculiar bluish or grey colour, similar to that which he used to have when the pressure was great in his head. He became dull and drowsy; the tongue deflected slightly to the right. When shown various things he could not name them, but could tell what they were for in the verbal form, *e.g.* a bandage he said was "for putting round head," and keys were "for opening doors." The pulse was 56 and soft. All pressure was removed. There was no sign of pus about the hernia. In the afternoon the pupils were unequal for a time. There had been no vomiting since the bandage was removed. On the 5th at 11 A.M. the pulse was 84. The patient was less drowsy, he answered questions more readily and gave evidence of amnesia of nouns but no word-blindness. He could read "Leith Monthly Visitor" fairly well, slowly but correctly. He was shown a picture of a railway carriage which he called "Eslanda." He called the flag in the guard's hand "thing to go round to go away," and the guard he called "man." He could not name a travelling trunk which was also in the picture. Some pus was found on dressing the wound. The hernia was larger. A drainage tube was introduced into the extra-dural space.—The amnesia of nouns was seen particularly in connection with the less familiar objects. He knew a safety pin, though he could not name it. He named a half-crown which is more familiar and often used; this may have been what Wyllie calls an "overflow" into the other hemisphere or partial education of it to common percepts. There was also "articulative ataxy"; he missed out parts of words—*e.g.* "wrying" for "writing."—On the 6th the patient's condition was similar. He was less drowsy. On the

8th the tongue was still deflected to the right; otherwise there had been much improvement since the drainage-tube was inserted. On the 10th the patient was very drowsy. During the night there was pain across the forehead. On the 11th and 12th the discharge was freer but it smelt badly. On the 15th although the patient's general condition showed improvement he could not even repeat the names of objects which were mentioned to him. When shown a key and asked to say "key" he said "exstrat"; when a knife was shown to him and he was requested to say "knife" he said "a maggie"; and on showing him a chain and asking him to say "chain" he said "mobelle." Therefore here word-intoxication and paraphasia are more marked. On the 19th he was examined by Dr William Elder. On attempting to read he could spell out the words and pronounce small words but could not read a simple sentence through so as to understand it. His memory for nouns was worse. He understood spoken speech quite well. He could not write his name but could write to dictation, A, B, C, D, &c. He was more irritable. Dr Elder notes: "This is probably due to spread of condition upwards. It is curious that no word-deafness was noted at this examination. Has the disease now spread towards the occipital lobe or angular gyrus?" On the 22nd the patient was not so well. He complained of a good deal of pain especially referred to the forehead. On the 23rd he had great pain and cried out a good deal. There were some spasmodic movements of the left leg. From the 24th to the 27th the patient's condition was getting worse. He was very drowsy and heavy. The pulse was slow, the temperature was subnormal, and he did not seem to understand when spoken to. The right grasp was absent and the left was very weak; the right knee-jerk was present and the left was absent. On the 28th at 9.30 A.M. there was Cheyne-Stokes respiration. The patient was unconscious. He swallowed with difficulty. There was no discharge. There were no localising symptoms. A pair of sinus forceps was passed forwards and somewhat upwards close to the bone and about half an ounce of thick green, very foul pus escaped. A drainage-tube was inserted. Immediately afterwards the pulse increased to 84; the respirations were full and regular. The reflexes could be got more readily. At 5.30 P.M. the patient was conscious, though deeply asleep

as a rule. He tried to speak but could only get out a jumble of words. On the 29th the patient was operated upon by trephining anteriorly to the hernia. The brain was very tense after the dura had been incised. No pus was got. At 10 P.M. the temperature was 105° and the pulse 142. The patient was quiet, sometimes moving a hand. On the 30th at 1.20 A.M. the pulse was thready and uncountable. The patient's breathing was laboured. During the day he gradually became worse and at 6.30 P.M. he died. From July 22nd onwards the signs had become more those of a general compression without localising symptoms. The second stage of the illness had shown an increase of the already existing paraphasia and amnesia of nouns, with some articulative ataxy, and finally death from compression took place.

A *post-mortem* examination was made on August 1st, 1896. The head only was examined. Behind the left ear was a hernia cerebri. This had been developed in the upper part of a vertical wound over the mastoid process. The hernia measured two inches by one and a half. From this a recent operation wound extended forwards in the temporal region. No pus was noticed in the ears. On removing the calvarium the dura was found to be congested and the convolutions were seen to be flattened. On the left side the dura was adherent to the brain over part of the lower surface of the temporal lobe in relation to the petrous portion of the temporal bone. There was a depression in this bone over the situation of the superior semicircular canal, to which the dura was adherent firmly by a small process of fibrous tissue. The hernia was connected with the posterior third of the middle and adjacent part of the lower temporal convolutions. A recent operation wound in the anterior part of the superior convolution was surrounded by extravasation into the brain substance which also extended to the adjacent parts of the ascending frontal and ascending parietal convolutions. There was laceration of the cortex in the inferior temporal convolution where the dura was adherent. In the posterior part of the inferior occipito-temporal and inferior temporal convolutions was a swelling at the part where normally is a hollow to receive the upper surface of the cerebellum. This swelling was due to extravasation of blood into the brain substance. On horizontal section an abscess containing thick green pus was opened into in the position of the left lateral ventricle. The surface was con-

gested, rough, and breaking down. The right lateral ventricle contained a small amount of non-purulent fluid. After hardening in formalin transverse coronal sections were made, and tracings were taken. The left temporal lobe contained an old abscess cavity in its anterior two-thirds, tailing off into the second and third convolutions posteriorly. Its contents were inspissated and white. The grey matter was thinned over it. The second (anterior) operation wound just stopped short of another recent abscess containing green pus. This was most extensive opposite the posterior part of the island of Reil. On section it was found to be in the position of the external capsule, and was invading both externally and internally, chiefly the latter, towards the lenticular nucleus. From this abscess a series of smaller abscesses extended backwards and downwards towards the base of the hernia, some reaching it. Internally and below, corresponding to the superficial swelling already noted, the lobe for about one inch in depth was infiltrated with blood. The hernia examined microscopically was found to be composed of breaking-down tissue infiltrated with pus, and into it by appropriate staining a few nerve fibres could be traced. The course of events had been : (1) middle ear disease, extending into the antrum, and affecting the facial nerve ; (2) extension by the superior semi-circular canal to the surface of the petrous bone, and thence to the dura mater and cerebrum ; (3) the formation of a primary abscess in the temporal lobe ; (4) a hernia cerebri ; and (5) secondary abscesses in the external capsule, and extending thence chiefly to the lenticular nucleus, and backwards towards the base of the hernia. These abscesses, with surrounding tumefaction and œdema, both general and in relation to the basal ganglia, accounted for the later compression symptoms.

Thus we have the illness commencing with middle ear suppuration, and probable direct extension to the temporal lobe in which the subsequent abscess formation affected the whole lobe, with the exception of the word-hearing centre, which was not affected at any stage. The abscess theoretically had interfered with fibres passing from below the word-hearing centre under the island of Reil to Broca's convolution, but was not at all in the line of those passing from the auditory centre proper to that motor speech area. The secondary abscesses were certainly in a situation which would interrupt these con-

necting fibres, but from the clinical aspects of the case we were disposed to connect them almost wholly with the more generalised symptoms of compression which went on to death, and which latterly veiled other localising symptoms. Probably, therefore, these secondary abscesses may be left out of account in explaining the aphasic symptoms.

The fact that the symptoms of aphasia and paraphasia were observed only after evacuation of the first abscess may be explained by the insensibility which existed for some time before the operation, and which masked a condition which only became evident when the causes of the coma were removed. After all, therefore, we may confine our consideration of the case to the primary abscess in the temporal lobe, and see how the clinical aspects of the case are borne out by the pathological findings.

We have, then, an abscess involving the whole white matter of the temporal lobe, with the exception of the auditory—word-hearing—centre. The aphasic symptoms noted were chiefly the paraphasia, of which the patient was in most cases conscious, and amnesia, particularly of nouns and names, while even in this it was noticed that the patient was able to name at times certain common objects. In connection with these articulative amnesia was noticed, parts of a word being given—*e.g.* "ence" for "pencil," "wrying" for "writing," and so on. With less familiar objects the patient was almost completely amnesic, though he could often denote by a sentence in the verbal form, or assisted by gestures, their use. The escape of the word-hearing centre explains his consciousness of the mistakes which he made, but the difficulty now is to explain his amnesia of nouns. The naming mechanism was at fault. The case seems to indicate that the area which controls this mechanism need not necessarily be identical with the auditory centre, but that there may be another part of the cortex in which memories of names are stored. No such centre has hitherto been described, though Elder in his book on Aphasia makes a suggestion which this case seems to support to a considerable extent. He says: "If, therefore, there is a naming mechanism separate from the auditory word centre, then the clinical and pathological evidence point to an important part of that mechanism being situated in the near neighbourhood of the auditory word

centre, but probably lower down—viz., in the posterior half of the third and second temporo-sphenoidal convolutions" (p. 136). "Amnesia verbalis, and especially the form where there is amnesia of nouns, such as proper names and concrete nouns, may occur, without there being any word-deafness or motor aphasia. Lesion in the temporo-sphenoidal lobe, especially if towards the posterior part of the lobe, produces amnesia verbalis; if in the second and third temporo-sphenoidal convolutions amnesia verbalis occurs without word deafness, but if the first temporo-sphenoidal is also involved the patient is word-deaf. These facts tend to show that an important part of the mechanism concerned in the recalling of words and names is situated in the temporo-sphenoidal lobe towards the posterior part of the lobe, and that this part of the mechanism is very closely associated, and probably continuous with the word-hearing centre, although not identical with it" (p. 139). "In connection with auditory aphasia it is necessary to remember also that amnesia of nouns may be a marked symptom, and in that case the naming mechanism is involved, an important part of which, as I have shown, is in all probability a little lower down than the auditory word centre—viz., in the second and third temporo-sphenoidal convolutions. If the patient showed difficulty in recalling names or in naming objects at sight without having word-deafness, the lesion would probably be found to be at a lower level in the temporal lobe than the first temporal, and if the patient showed a marked difficulty in naming objects at sight, probably the lesion would be subcortical or deep in the substance of the posterior part of the temporal lobe" (p. 251).

Now, admitting the existence of such an area, the conducting fibres from it to Broca's convolution would be interrupted, and the results would be such as were found in this case. Certainly all depends upon the elimination of the other factors present, and we think that they may be eliminated on very reasonable grounds, as already detailed.

## Meeting V.—February 1st, 1899

Sir John Batty Tuke, *President, in the chair*

### I. Exhibition of Patients

1. *Dr R. A. Fleming* exhibited a mother and family of four children apparently suffering from Addison's disease.

The mother, a spare woman, is 28 years of age, and has come of a fair-skinned race, although her hair is dark, and there is no tubercular family history. The mother's mother and mother's sister both died of cancer, otherwise the family records are irreproachable, and both the patient's father and mother are alive and healthy. Previous to her marriage eight years ago, she was delicate, but not pigmented in any way. With her first pregnancy she became somewhat darker than usual, and with each successive pregnancy, five in all, the pigmentation increased. Two years ago her youngest child was born, and not merely was she darker than before, but she became more languid and less able for her ordinary house work. When seen for the first time by Dr Fleming in July of last year, 1898, she was found to be deeply pigmented in the usual sites for pigmentation in Addison's disease, with the exception of the mucous surfaces of the mouth which were only slightly coloured on the inside of the cheeks.

The eyes, nipples, axillæ, and abdomen were specially dark, and numerous black pigmented spots or moles were seen on the face, limbs, and trunk. These have increased in number during the past six months. The skin is somewhat darker in the summer than in winter, but is steadily becoming permanently darker in colour. The patient has had several typical attacks of nausea and vomiting, and many marked attacks of diarrhœa without any definite or apparent cause, and has suffered from repeated faint turns, much giddiness, and frequent attacks of palpitation. Pain in the back is fairly constant, and the languor and debility have greatly increased during the past year, although there has been some improvement under treatment.

The oldest of the family, aged 7 years, a girl, has dark hair and deeply pigmented skin. The nipples are dark. She was a fair-complexioned child until four years ago, when, after an attack of gastric catarrh, the mother says she became somewhat

sallow. Pigmentation has greatly increased lately with the appearance of numerous moles, she has had attacks of causeless nausea, vomiting, and diarrhœa, and from being a sprightly child has become languid and weak.

The second girl, aged 4 years, was a fair child, has fairer hair than her sister, and has only become dark a year ago. The nipples are slightly pigmented, and there are numerous moles. She too has had similar attacks of nausea, vomiting, and diarrhœa, and is more languid and less inclined to play than formerly.

The third child is a boy, aged three years. He has always been dark-complexioned, but for the last six months has become much darker, and a number of pigmented moles have appeared during this period, and are still appearing. He has had several attacks of vomiting and diarrhœa, but not so severe.

The fourth child is a girl, aged two years. She was fair, and is only within the past six weeks becoming slightly darker, and three weeks ago two pigmented moles have appeared.

One child born several years ago died in infancy of bowel complaint.

The mother has been treated with arsenic, strychnine, &c., and has somewhat improved. She has not yet been given suprarenal extract.

Dr Fleming stated in showing these cases that if his diagnosis of Addison's disease was correct, the family group constituted a unique instance of the condition.

2. *Dr William Russell and Mr J. M. Cotterill* exhibited a case after operation for MASTOID DISEASE and EMPYEMA. *Mr Cotterill* said the case was one in which a remarkable recovery had been made from a condition of mastoid abscess spreading along the lateral sinus into the posterior fossa of the skull, and down along the vein into the upper part of the neck. Mr Cotterill deprecated the use of an exploring syringe in order to diagnose the condition of the inside of the lateral sinus. This was not devoid of risk, and was unnecessary as the diagnosis could be made by palpation and pressure below upon the vein. The fact that empyema in this case was secondary to the mastoid abscess was contrary to the usual rule.

## II. Exhibition of Specimens

1. *Dr T. Shennan* exhibited—

(*a*) Early tuberculosis of the urethra as it passed through the glans penis—microscopical specimen—from a case of genito-urinary tuberculosis.

(*b*) Microscopical specimen of the smaller vessels from a case of tubercular meningitis, showing proliferation of the endothelial cells, lining the perivascular lymphatics.

(*c*) Similar organisms from a case of cancrum oris, and a slow spreading gangrene succeeding a bruise of the arm. It resembled the involution form of the diphtheria bacillus. It was separated by means of agar-agar, was non-pathogenic in pure culture, and though sub-cultured frequently retained its peculiar shape, that of a beaded bacillus with clubbed ends.

2. *Dr R. A. Fleming* exhibited—

(*a*) Two mammillated stomachs. (1) One was that of a woman, aged 54, who had been under treatment for a considerable length of time.

The stomach throughout was thickened, and the small projections on the mucosa covered the whole stomach wall. The patient had died eventually of persistent vomiting, and consequent exhaustion, notwithstanding administration of the usual remedies.

(2) Was the stomach of a man, aged 40, who had suffered for many years from heart disease with backward pressure. There was less interstitial thickening, but the same extensive mammillated appearance of the mucosa. Persistent vomiting was a marked feature of the last few weeks of life.

(3) A third stomach was exhibited for the purpose of shewing how a certain degree of mammillation was common in cases of chronic gastric irritation. In this case a large chronic simple ulcer had formed and was firmly adherent to the liver, which had been perforated for some distance in the floor of the ulcer. The pyloric region of the stomach was markedly mammillated.

Dr Fleming pointed out that mammillation was not uncommon in cases where such chronic irritation existed in the stomach, that it was most common near the pylorus, and was often associated with thickening of the pyloric end of the organ, though sometimes it occurred in a dilated and otherwise

attenuated stomach. In the first two cases the extensive distribution of the condition was associated with an uncommon degree of gastric irritability.

(*b*) A specimen and sketch of an aneurism of one of the sinuses of Valsalva which had almost perforated into the left auricle. The condition was associated with ulcerative endocarditis of the aortic cusps and was obtained from a man who died at the age of 33.

3. *Dr William Stewart and Dr John Thomson* exhibited a CONGENITAL MALFORMATION OF THE ŒSOPHAGUS.

This specimen was taken from a male child, the sixth in a family of well-developed, healthy children. It was born on the evening of the 28th December. The labour was tedious but easy. The mother had had a fall downstairs at the fourth month.

The child was born semi-asphyxiated, and required the removal of a large quantity of mucus from the pharynx, and more than the usual number of "slappings on the rear" to induce crying.

Next morning the child still looked blue, and did not breathe satisfactorily; and he (Dr Stewart) was told that it had had choking fits during the night, and did not swallow the milk and water, with which an attempt at feeding had been made.

It had passed meconium of natural appearance, and had made water.

On attempting to feed it with sweetened water it was observed that the movements of swallowing were accomplished in a natural manner, but that after it had had about a teaspoonful, it became blue in the face, and had to be turned over in order to allow the water to run out of the pharynx. It fastened on the breast however, and the mother stated that she was sure the child had swallowed and retained some milk; thus, though an obstruction, causing difficulty of swallowing was diagnosed, it was hoped that the child might have got some small quantity down. Next day, the ability to swallow not having increased, a soft india-rubber nasal tube was passed through the mouth down the œsophagus, but was arrested at a point 5 inches from the mouth. The passage of a medium-sized india-rubber catheter showed the same result. Enemata of wine whey and brandy were given.

On the following day the child was seen by Dr John

Thomson, who, having passed a tube which was stopped at the same point, confirmed the diagnosis of malformed œsophagus, either atresia or a transverse septum.

The child seemed then so weak, besides having crepitations over both lungs, that it was thought better to leave him alone.

However, on the morrow (1st Jan.) the child looking better, and the lungs having to a great extent cleared up, it was thought advisable to give him the chance of operation.

He (Dr Stewart) therefore, with the assistance of Major Mitchell and Mr Miles, did a gastrostomy after the manner of Witzel.

The little patient stood the operation well, and after having been fed through the tube with about six teaspoonfuls of wine whey mixed with a little brandy, looked better than it had done for a couple of days. He saw it two hours afterwards and again fed it.

Its colour and pulse were then good, but he was hurriedly sent for in about an hour, and on arrival found the child dead.

He was told that it had had retching, and then became blue and died.

The explanation of the immediate cause of death will be seen in the specimen which shows a communication between the stomach end of the œsophagus and the trachea.

The other organs were healthy and normally developed.

At the *post-mortem* examination the following condition was found :—

The upper part of the œsophagus ended in a rounded extremity about 1 cm. above the bifurcation of the trachea. It measured 1 cm. in diam., and its walls were much thickened, apparently from muscular hypertrophy. There was no communication between this part of the œsophagus and the stomach, and it did not communicate with the air passages.

The stomach was normal in size and shape, the wound near its pyloric end was very healthy. The œsophagus seemed of normal calibre at its lower end, but, when followed up, it was found to become narrower and to open into the trachea at its bifurcation, the opening being large enough to admit a No. 2 catheter. A small bundle of muscular fibres from its posterior wall passed up the back of the trachea, and seemed to become continuous with the anterior wall of the upper cul-de-sac. The stomach was full of fluid food, and the immediate cause of death

had evidently been the vomiting of some of this into the air passages.

The lungs, when cut into, smelled strongly of stomach contents, and traces of food were visible in the bronchi. There was pneumonia of both bases. The other organs were normal, so far as examined.

4. *Mr J. M. Cotterill* exhibited a piece of the TRANSVERSE COLON removed after death from a patient upon whom he had operated eleven years before, resecting upon that occasion no less than twenty-two inches of colon, which had become strangulated and gangrenous in the sac of an umbilical hernia.

Though some 150 silk stitches had been put in on that occasion, the upper and lower segments of gut which were united being of very different sizes, and so leading to great difficulty in joining them by Czerny Lembert Sutwing, there was now great difficulty in recognising the exact line of suture. The patient had lived comfortably for eleven years, and died of another abdominal complication.

## III. Original Communications

### 1. THE TREATMENT OF OZÆNA WITH SPECIAL REFERENCE TO CUPRIC ELECTROLYSIS

By P. M'Bride, M.D., F.R.C.P.Ed., F.R.S.E., Surgeon, Ear and Throat Department, Royal Infirmary; Lecturer on Diseases of Ear and Throat, Edinburgh University.

As the term ozæna has been used in a somewhat vague manner throughout medical literature, I may perhaps be permitted to give a very brief definition of the disease as it is now understood by specialists.

The affection is characterised by atrophy of the nasal mucosa, and sometimes even of the osseous structures of the turbinateds. This atrophy depends upon (1) fatty degeneration of the glands, followed by disappearance of these structures; (2) fibroid changes in the mucous membrane. At some period —some say from birth—the ciliated epithelium changes its character and becomes flat and horny, the ciliæ of course becoming lost. As a result of these alterations, there is a tendency for the nasal secretion to dry up rapidly when brought

into contact with air, and to form greenish crusts which emit the horrible stench so familiar to all who have met with cases. It is of importance to note that the fœtor is not fully developed until the secretion has dried. This can be easily demonstrated by cleansing the nares and then plugging with absorbent wool. The latter, if left in for some hours, is found to be soaked with fluid secretion, but often almost devoid of odour.

On examining the nares of a patient afflicted by ozæna, after removal of the crusts, no ulceration or lesion of any kind will usually be detected, excepting the marked atrophy which is commonly most noticeable in the inferior turbinateds.

Of late years numerous organisms have been detected in the secretion. Thus Löwenberg, Hajek and Paulsen have each described bacilli, but in no case, so far as I am aware, has one been found capable of reproducing the disease. Belfanti and della Vedova have, as a result of their investigations, ascribed ozæna to an organism closely resembling, if not identical with, the Löffler bacillus of diphtheria, but have adduced little more definite proof than the other observers referred to.

The prognosis of ozæna is, as every practitioner knows, not satisfactory. The affection usually begins about puberty, and sometimes towards middle life the worst symptom—viz. the fœtor—disappears, while the atrophy persists. Under continued local treatment the odour may often be masked, and until recently this was considered the best result capable of being achieved.

It would serve no useful purpose to discuss the various remedies which have been proposed for this all but incurable malady. Indeed, to do so would occupy much time without yielding any satisfactory results, for as in all intractable affections so in ozæna, innumerable drugs have been advocated. On the other hand it will be well to glance briefly at the therapeutic principles which have been invoked, and to consider very shortly some of the methods which have been utilised in carrying them out.

(1) *Destruction of the secreting area.*—It was many years ago proposed to destroy the lining membrane of the nose by means of scraping instruments, but this method has not to any appreciable extent survived the criticism of Voltolini, who, in his great work ("Die Krankheiten der Nase," Breslau, 1888), writes: "I never employ scraping with a sharp spoon; I leave that to others, and observe what effect they have produced.

Thus I have observed that it does absolutely no good, and that the nose rather looks worse than before," &c.

(2) *The application of stimulants and irritants.*—The employment of tampons of wool as suggested by Gottstein is an efficient palliative, as by their use fœtor is reduced. It is, however, difficult to get patients to tolerate plugging of the nostrils for a sufficient length of time. The application of irritating liquids, such as spirits of mustard, and caustics of various kinds has been much recommended, and good results have been especially attributed to spraying with a concentrated solution of nitrate of silver, as suggested by Meyjes. Mechanical stimulation has also been advocated. Thus Fränkel suggested light touching with the electric cautery, while Braun, Laker, and Demme have advised vibratory massage of the mucous membrane. This is carried out by introducing into the nostril a probe surrounded by cotton wool which may be saturated with a suitable medicament. By bracing the arm the instrument is made to vibrate rapidly. As the exertion required has been found somewhat fatiguing to operators, electric motors have been suggested as substitutes for the hand.[1] This treatment seems really to have been attended by success, and Schech states the micosa may thus not only be brought back to the normal, but even caused to become hypertrophised. Chiari, on the other hand, ascribes the benefit derived to the irritation and to the solutions usually employed at the same time. He has obtained good results by painting the interior of the nose with Mandl's solution. The valuable effects of this last-named treatment I can fully confirm.

(3) Since the widening of the nasal channel which results from the atrophic condition, tends to make elimination of secretion difficult, it has been proposed to use apparatus for producing partial occlusion—so-called nasal obturators.

(4) As remedies which do not come exactly under any of the above headings, I may mention a spray containing borax and 70% of glycerine as suggested by Musebold, and ichthyol in solution as advocated by Eitler, who uses the drug for syringing (2-5%), and painting (25-30%).

All these methods have been employed, some of them have yielded a degree of success, but still the fact remains

[1] "Krankheiten der Mundhöhle des Rachens und der Nase." Leipzig und Wien, 1896, p. 297.

that in many quarters careful cleaning of the nares by means of alkaline, antiseptic, and other solutions, is looked upon as the most satisfactory treatment which can be instituted.

Of late two novel and strangely divergent lines of treatment have been advocated—to wit (1) the injection of diphtheria antitoxin; (2) cupric electrolysis.

As already stated, Belfanti and della Vedova[1] believed that they had found an organism in ozæna which was practically identical with the Löffler bacillus. They therefore employed antitoxin in thirty-two cases and recorded sixteen cures and more or less improvement in all the others. Gradenigo[2] to a considerable extent confirmed the statements of these two authors, *i.e.* he found that benefit and even cure resulted from the injections; he, however, soon after advised intra-muscular injections of iodine as a substitute. Arslan and Catterina[3] also experimented with antitoxin and admitted that it had a considerable effect on the mucous lining of the nose. They found the injections often followed by epistaxis, the mucosa became more moist and vascular and even the odour was modified, but all the improvement rapidly disappeared. Another observer—Compaired[4]—found marked benefit so far as the local affection was concerned, but the repeated injections produced fever and rashes.

I think that from the above observations we must deduce that a very definite although probably temporary effect is produced by antitoxin. I may, however, say at once that I have never employed it because it seems hardly fair to subject patients to prolonged injections of a powerful remedy of this kind which may be far from harmless, unless much more convincing proofs of its efficacy can be adduced.

## *Cupric Electrolysis.*

According to Moure, Garrigon-Désarènes[5] was the first author who proposed the employment of electrolysis. He, however, applied only one pole to the surface of the nasal mucosa, while the other rested upon the neck or some other

[1] "Rev. Hebd. de Laryngologie," &c. 2 Mai 1896.
[2] "Annales des Mal. de l'oreille," &c. Aug. 1896.
[3] "Rev. Hebd. de Laryngologie," &c. 17 Oct. 1896.
[4] "Annales des Mal. de l'oreille," &c. Mai 1897.
[5] "Bulletins de la Soc. Franc. d'Otologie," &c., xiii. p. 1.

indifferent part. His method was followed in a more or less modified manner by Delavan of New York. Then Jouslain suggested interstitial cupric electrolysis in an article which unfortunately has not been accessible to me but which Moure (*op. cit.*) states was communicated to a French scientific society in 1892. This method of treatment was not apparently referred to again until 1895. Cheval described it and at the same time recorded 91 per cent. of cures, and in seventy of his cases attained this result after a single séance. His paper was communicated to the Society of Belgian Laryngologists and Otologists,[1] and later this body appointed a committee of four to enquire into the effects of cupric electrolysis. Seven cases were treated by Cheval and in none of them could the members find any improvement. In 1896 Bayer[2] gave a long account of the method and its results, which according to him were extremely good in a large proportion of cases. During the following year Réthi[3] expressed the opinion that the benefits obtained from cupric electrolysis were greater than those derived from any other method of treatment.

Brindel,[4] at the suggestion of Moure, undertook the treatment of thirty cases in the clinique of the latter. He found that in every instance the nasal affection was modified for some days; the mucosa became swollen, red and congested; the yellowish green thick crusts no longer formed on the side operated upon; in their place sticky mucus sometimes tinged with blood was found while fœtor disappeared. Brindel, however, observed that relapses often occurred after from one to eight weeks. Nevertheless, in ten cases he found permanent benefit to have followed the treatment. In none of these at the time of writing were there either crusts or odour, the patients having been under observation from three to eleven months.

The latest work upon cupric electrolysis with which I am acquainted emanates from Gouguenheim and Lombard,[5] who arrived at the following conclusion, viz. cupric electrolysis has a definite action on ozæna, causing disappearance of fœtor

[1] "Centralblatt für Laryngologie." 1896. P. 406.
[2] "Rev. Hebd. de Laryngologie." Mai 1896.
[3] "Wiener Klin. Rundschau." No. 10. 1897.
[4] Moure *op. cit.*
[5] "Annales des Mal. de l'oreille," &c. Nov. 1898.

but it is not possible to say that the result will be permanent.

I do not think that cupric electrolysis has yet been touched upon in English literature, and with one exception its use seems to have been so far confined to Belgium and France. Struck by the preponderance of favourable evidence I began to experiment with this treatment in 1897 and shall give my results as briefly as possible.

(1) W. MacD., æt. 26, labourer, conscious of having had nasal trouble since ten years of age, odour offensive to friends, no sense of smell, great atrophy of inferior turbinateds with marked crusts. Electrolysis performed five times. The first séance was in October 1897, the second a month later and the others at intervals of a fortnight. He at once began to improve, the smell disappeared and the secretion became liquid.

When last seen (13th Dec. 1898) he had done no syringing but had occasionally to clear the nose; there was however no bad smell. On examination a somewhat moist detached crust was found in the right nostril while the mucosa on the left side was clean and moist. The parts were still atrophied. A good deal of fluid secretion was present in the naso-pharynx.

(2) K. M., a girl of 17, seen first in 1897, suffering from crusting, fœtor and atrophy. During eighteen months has had four sittings, has never syringed during this time.

Dec. 13th, 1898. Keeping well; no fœtor; occasionally something on handkerchief which comes away easily.

Objective. Right nostril; small non-smelling crust on floor; some moisture; middle turbinated slightly glazed. Left nostril; general moisture.

Atrophy remains in both; naso-pharyngeal secretion causes inconvenience.

(3) R. J., female, æt. 20. Seven years of crusting and fœtor; no subjective sensation of smell; crusts very adherent.

In November 1897 electrolysis was employed for left nostril, and repeated twice afterwards; smell diminished but did not cease; crusts came away more easily and there was fluid discharge from nose. About six months afterwards she noticed that the condition was again worse but it has never been so bad as before the first séance.

In November of last year (1898) electrolysis was again employed for left nostril.

When last seen, on 15th December 1898, it could hardly be said that there was appreciable improvement as the patient had to syringe twice daily to prevent crusting.

This patient was again seen on the 24th January 1899. She was told not to syringe and on the 26th there was no fœtor but some moist crusts were seen both in the anterior and posterior nares.

On the 31st there was no fœtor but crusts had accumulated in the naso-pharynx and larynx, although hardly at all anteriorly.

(4) C. C., female, æt. 24. When first seen had suffered from bad smell and crusts for 10 years. The inferior turbinateds here were not so much atrophied as in the other instances but the case was still a pronounced one.

Cupric electrolysis was used in the right nostril on the 25th July 1897; on the 27th the condition was much improved, no more crusts, odour hardly perceptible, general moisture excepting on middle turbinated, while a good deal of fluid muco pus was seen lying about.

1st August 1897. Electrolysis again employed, this time in left nostril.

7th August 1897. More crusting, very slight odour, electrolysis again.

Examined for the last time, 12th December 1898. She then stated that she relapsed two or three weeks after last sitting and she thought she was very much as before treatment, requiring to use the syringe daily.

(5) A. D., female, æt. 30. For more than two years suffered from considerable crusting and offensive odour—marked atrophy of inferior turbinateds.

July 25, 1897. Cupric electrolysis applied to right nostril.

July 27. Marked improvement in both nostrils—no odour—no crusts—general moisture and some fluid muco-purulent secretion.

August 27. Electrolysis applied to left nostril—no syringing for a fortnight—no odour or crusts.

Examined 10th December 1898. Keeps very well; during the preceding four months had only required to syringe the nose once. There was a little discharge from the nose.

(6) M. M., female, æt. 22. History of crusting and fœtor for

six years—atrophy of inferior turbinateds—a bad case. Cupric electrolysis was first employed in June 1897 and was repeated every three weeks until six sittings had been accomplished. Patient writing on December 14, 1898, stated that she had never syringed her nose since the treatment, that the odour had never returned, but that a few crusts still continued to come away.

(7) J. B., male, æt. 19. Had crusts and fœtor for 5 or 6 years—had syringed for some years.

In May 1898 electrolysis was used for left nostril, and a fortnight later the right nostril was treated. A third and fourth sitting followed. The trouble disappeared until the middle of September when fœtor and crusts recurred and the patient resumed syringing which had been given up. On the 27th December 1898 he was very much as before treatment was begun.

(8) J. B., female, æt. 20. Crusts, fœtor and atrophy for one year (?). She also complained of feeling bad smell and taste. No previous treatment; electrolysis October 1898. When examined in January 1899 felt much better—no perceptible odour but slight crusting and persisting atrophy.

Although a much larger number of cases were treated, I have been unable to keep records of them owing to the sufferers having been seen as out-patients, and also because I have not a sufficient staff of clerks. Indeed I am very much indebted to my friend, Dr Logan Turner, who kindly kept the records I have just given. It may, however, be permissible to state that the cases given were in no sense selected. The strength of current used varied from three to ten milliampères, rarely exceeding the latter. Cocaine was used in most, if not in all, and after cleansing the nostrils the copper needle attached to the positive pole was inserted into the inferior or middle turbinated—sometimes into the tissues lining the middle meatus, while the platinum (or steel) needle was passed into the septum. Usually the patients complained of little pain nor did they experience disagreeable after effects. A private patient on whom I recently performed electrolysis, however, suffered a good deal from neuralgia, swelling of the eye and general disturbance for a day or two. As a rule each sitting lasted about 10 minutes. As I have said before, the cases quoted were not selected by us but it is just possible, nevertheless, that a process

of natural selection may have been brought about. They all occurred in out-patients, and it is quite conceivable that the majority of those who were benefited repeatedly presented themselves, while there may have been an opposite tendency on the part of those who received little relief. It will be noted that out of the eight patients whose histories have been briefly given, four (1, 2, 5, 6) were practically cured for long periods extending to 18 months; in one (8) there was marked improvement; in one (7) apparent cure for some months, but then syringing had to be resumed; while in two (3 and 4) there was only improvement for a few weeks; the former is here to-night and it appears to me that the fœtor has been practically cured but that what she complains of is the discomfort caused by crusts accumulating in the naso-pharynx and larynx.

In the last sentence, when I use the term "cure," I refer to the fœtor, for in most of the cases the atrophy remained as before. I am quite prepared to admit, as above stated, that the results achieved in these seven cases may have been beyond what we can reasonably expect to average. Still they prove that in cupric electrolysis we have a valuable therapeutic resource—probably the most valuable that has yet been suggested in ozæna. The rationale of the treatment is still uncertain, but probably the formation of copper salts at the positive pole has at least as much effect as the electric current.[1]

## 2. ON MOVABLE KIDNEY

By David Wallace, F.R.C.S.Ed., Assistant Surgeon, Royal Infirmary, Edinburgh

Movable kidney is the term used to designate a kidney which moves externally to the peritoneum through a greater area than the kidney does normally. It is a relative term, as the kidney normally moves to some extent during respiration. In thin persons with lax abdominal walls the kidney can be felt to move, but its movement can be best estimated in those cases where, for purposes of treatment, the operation of nephrotomy has been performed. We then find that the kidney moves down

[1] It will be noted that the application of electrolysis to one nostril in our patients often produces benefit, temporary or otherwise, on both sides. This is in accordance with the experience of most other observers.

with inspiration and upwards with expiration, while it is tilted forwards on an antero-posterior axis.

The condition of movable kidney, in the sense referred to, has been known as an abnormal condition for many years—the first reference to it being as far back as 1581, while its clinical importance was set forth by Riolau in 1649. King in 1836 attempted to excise a movable kidney but failed to find it after opening the abdomen. We may take it however, that, in spite of such early pioneers in this branch of surgery, the importance and significance of movable kidney were not recognised until much later—not until 1860, and indeed for twenty years after that date little was done from the clinical standpoint. Prior to 1880 Keppler, who believed the condition to be dangerous to life, advocated excision of the kidney, but this heroic procedure was adversely criticised, and when in April 1881 Hahn of Berlin proposed, and carried out, stitching of the organ to the posterior wall of the abdomen, excision was discarded, and nephrorraphy or nephropexy, as it is now termed, took its place.

The subject up to 1881 is very thoroughly discussed by Landau in a monograph which appears in vol. 110 of the New Sydenham Society. It is still further brought up to date by Dr Newman of Glasgow in his work "Diseases of the Kidney," 1888; and in all its bearings, except operative, it is in the most complete manner considered by Dr Fischer of Vienna in the *Centralblatt fur die Grenzgebiete der Medizin und Chirurgie*, 1898. Dr Fischer further gives a most elaborate bibliography of the subject at the end of his paper. In consideration of these works being readily accessible it is unnecessary for me to occupy your time by a discussion of the statistics, etiology and symptomatology of movable kidney, but I propose to bring before you some points which have been more recently advanced regarding the etiology, and to illustrate the subject from cases which have been under my personal observation and care.

As regards the frequency with which mobility occurs, I believe *post-mortem* statistics are fallacious, as the kidney is much more fixed after death than during life. Clinically the condition is not uncommon, although at the present time authors, I think, rather exaggerate its frequency. It occurs more frequently in women than in men, the proportion usually being given as 10 to 1. The right kidney is more frequently affected

than the left, the proportion here being also 10 to 1. Why this should be no satisfactory explanation has been given in my opinion, and indeed the causes assigned for the mobility seem to me erroneous and inadequate. It is said to occur chiefly in those women of the poorer class who have borne many children at short intervals, and who have been unable to stay in bed for a sufficient length of time after parturition. Greig Smith says, "a considerable proportion occur in the upper classes, and most of these have been addicted to violent exercises—riding, tennis and gymnastics. Of those that come under the observation of the surgeon for treatment, most are to be found in this class." This coincides with my own experience. Of 14 cases requiring treatment 13 were females, and of these 7 were nulliparous, and only 1 of the others had borne several children and belonged to the poorer class. In a recent paper, January 1899, *Annals of Surgery*, Dr A. B. Johnson gives expression to a similar opinion and quotes cases treated in the Roosevelt Hospital, New York, to bear it out.

Then again it is stated that loss of fat predisposes to the condition, but I am inclined to agree with Senator that loss of fat may merely render the condition more easy of detection, and I am sure that in some cases, those in which gastro-intestinal symptoms and neurasthenia are present, the emaciation is a result not a cause. So too I think the view that mobility is most common in multiparæ may be due to a similar fallacy, viz., that the abdominal wall in such women is more lax, and the kidney therefore is more easily palpable than in nulliparæ. I believe of one thing we may be satisfied: the condition comes on gradually in the majority of cases and gets progressively worse; while the exact time of its onset is rarely known unless it be due to traumatism. Attention is now being drawn to the occurrence of movable kidney in children, but it is stated that in them it may be readily overlooked, as palpation of the abdomen, except under an anæsthetic, is difficult. Recently Dr Comby has written upon this subject in the *Archives de Médecine des Enfants*, 1898.

Recently much work has been done in reference to the shape of the kidneys. Dr Cunningham of Dublin and others have demonstrated that their shape is not merely that of the ordinary description, but that each is moulded by both the solid and hollow viscera in relation to which they lie, and that

the twelfth rib, the psoas muscle and the crus of the diaphragm mould their posterior aspect. The anterior surface is in particular at variance with the usual description—upon it "there is a point of maximum convexity—a place where the kidney substance is raised in the form of a marked prominence or bulging which in the right kidney extends across the anterior surface in the form of a rounded ridge. Above and below this eminence the anterior surface falls away towards each extremity in the form of an inclined plane of greater or less obliquity. These planes represent the effect of pressure. The superior from the liver, the inferior from the ascending colon. The intervening eminence is the result of this counter-pressure. Frequently the inferior sloping surface presents a high degree of obliquity." The moulding described by anatomists does not invariably present itself, as can be seen *post-mortem* — the kidney being examined *in situ*—when, in many cases, the kidney has the smooth convex anterior surface described in the older text-books. Further, the moulding when present varies considerably from the above description. Can it—the moulding—be due to *post-mortem* changes from pressure while the body lies on its back? It would be interesting to harden with formaline, as it is now used, the body being in the erect position or lying on its face. Dr Cunningham also alludes to the "clasping" action of the twelfth rib as manifested by the moulding of the kidney posteriorly. Is this also due to the recumbent posture after death, or is the twelfth rib during life an important aid to fixation of the kidney?

Kendal Franks (*Brit. Med. Journ.*, 1895) draws attention to this shape of the kidney and propounds the view that displacement is in great part prevented by it, and suggests that too great mobility occurs when the upward pressure is in some way diminished. He suggests two causes which may give rise to such diminution of pressure. 1st, parturition, 2nd, traumatism; but both of these causes are absent in many patients. Negative pressure, so called, after parturition, if it obtain at all, probably is only present for a few hours. We have an analogous condition after ovariotomy when if a large tumour be removed from the abdomen pseudo-ileus may supervene, but quickly passes off. Laxity of the abdominal wall, after parturition, is another matter, and may diminish the support of the kidney, but *negative* intra-abdominal pressure does not accompany it.

So far as these explanations are concerned I think they still leave us in doubt regarding the cause which initiates movable kidney, and I believe we must seek for the explanation in variation of anatomical relationship and fixation. Perhaps, as Greig Smith indicates, normal mobility may be a source of stability, and abnormal mobility may result from too great original fixation. Why, however, the right kidney should be more liable to the condition than the left, and why it should be more common in women than men, still remain a mystery. I cannot agree that tight lacing and the liver account for these facts, and in any case many instances are on record where tight lacing has not been indulged in, and where the left kidney has been the one affected.

The detection of movable kidney is not as a rule difficult, but occasionally a kidney which is freely movable at one time is not readily felt at another. Sometimes we can displace the kidney by adopting some such method as Greig Smith recommends, when we fail to get it in the usual way. When palpating the lumbar region for the kidney we may fail to find it because it is lying, at the moment, low down in the iliac fossa —this is a source of error which should be borne in mind. I have in two instances discovered the kidney—which had been previously overlooked—lying in this position: in one of these an error of diagnosis had arisen, the kidney swelling being supposed to be a swelling associated with appendicitis. Another error into which we may be led is the idea that the freely mobile kidney is enlarged—this, I believe, is due to a physical deception, viz., the kidney gliding under the hand seems to be larger than it really is. I have operated where the kidney was supposed to be of large size and found a kidney of normal size and shape. Once we feel the swelling recognition is generally easy, as 1st, it may have the general kidney shape, and we may feel the hilus; 2nd, it slips back into the kidney region; 3rd, the character of its movement is distinctive, "like a cherry stone from between the finger and thumb," as Landau says.

From the clinical standpoint the symptoms associated with mobility may be grouped under four heads. 1st. Simple mobility which gives rise to no symptoms. 2nd. Pain which is in no way characteristic of a renal affection, and is described as a sensation of dragging and aching on the affected side.

3rd. Renal pain distinctive of renal conditions. This may be accompanied by hæmaturia, pyuria or intermittent hydronephrosis, but not necessarily so. 4th. Gastro-intestinal symptoms which may be accompanied by, or end in, neurasthenia. It must be remembered, however, that the symptoms are not always commensurate to the degree of mobility. Some extremely mobile kidneys give rise to no symptoms, while less mobile kidneys may cause most aggravated symptoms.

The affected kidney is usually healthy. Thus in those excised prior to the introduction of nephropexy the majority were perfectly healthy. No doubt in some instances the kidney contains a calculus, or may be the seat of tubercle, or may have changes due to pressure associated with intermittent hydronephrosis, yet in the majority of cases there is no sign of disease. In eight cases upon which I have operated only one exhibited any abnormal condition, and in that case intermittent hydronephrosis had been present for two years. In that case the kidney pelvis was considerably dilated. In one case in which, after failure (?) by nephropexy, I excised the kidney it was small in size, but apparently quite healthy. [The patient in this case suffered great discomfort, and was practically an invalid ; for two years before operation she was never able to be out of doors. After nephropexy she had relief, but gradually her old complaints reasserted themselves, and I thought nephrectomy justifiable and performed that operation rather more than a year from the date of the nephropexy. At the second operation I found the kidney firmly fixed by a broad band of short fibrous tissue, so firm that I required to cut through it with scissors. The patient improved markedly, and is naturally free from pain.] Even in those cases which suffer from symptoms such as we associate with renal affections the kidney is usually healthy. In some calculi have been got, but only in a minority. The symptoms wholly subside as a rule after fixation of the kidney.

The explanation of the gastro-intestinal symptoms given by Kendal Franks seems to be reasonable, and the fact that such aggravated symptoms are recovered from after operation seems to verify his opinion. He points out 1st, that some symptoms are common to both kidneys ; 2nd, that some belong exclusively to mobility of the right kidney ; and 3rd, that some occur in mobility of the left kidney. In the first category he places the dragging pain, feeling of weight, and neuralgic pains in

the loins, fatigue on slight exertion and general debility. In the second group he places those symptoms recognised as "gastric crises," which are in effect gastro-intestinal: indigestion, flatulence, vomiting and pain, which come on about two hours after food. Relief from these may be got by resting in the recumbent posture. The second or descending portion of the duodenum lies in relation to the right kidney and rests partly upon it. It lies behind the peritoneum and is fixed very much as the kidney is. Kendal Franks believes the explanation of such symptoms to be that the kidney gradually drags the duodenum down and, as he observed in one case upon which he operated, produces a kink upon it, so that a temporary condition is induced, very similar in its symptoms to what is found in pyloric stenosis. I do not suppose that the drag on the duodenum is such that in all cases a kink is produced, but from a lesser degree of displacement similar symptoms though not so extreme may result.

Intermittent hydronephrosis due to movable kidney is not of very frequent occurrence, but when met with the history is fairly definite of a swelling in the lumbar region, usually felt anteriorly, which comes and goes and occasionally attains considerable dimensions. When the swelling is present the kidney is indistinctly felt, and may appear fixed, but when the swelling disappears the kidney is readily felt to be movable. Dr Knight, in a thesis presented for graduation as M.D. to the Aberdeen University in 1893, discusses the subject in detail, and gives a table of 111 cases. Of these, in the first 31 cases quoted—up to the year 1873—only 2 are said to be due to movable kidney. While of the remaining 80—from 1876 to 1893—47 are stated to have been associated with mobility. Of the remaining 33 cases in the majority no cause is given. Fixation of the kidney may get rid of the condition which is probably due to a kink of the ureter, but when the pelvis of the kidney is much dilated and the ureteral opening into it is much changed in position, or when valvular folds form, as described by Mr Henry Morris, further operative measures may be required. In the lecture which deals with this subject, which Mr Morris has recently published in "The Origin and Progress of Renal Surgery," he brings this aspect of the question up to date, and shows that such cases may be cured by ureterotomy, and that nephrectomy which used to be frequently recommended may

be dispensed with. Probably the valvular conditions of the ureter, or in the pelvis of the kidney, are in many cases results, not causes, of the hydronephrosis.

In the work by Mr Morris alluded to, statistics of nephropexy from his own cases are given in tabular form, and I may shortly quote these before I refer to those cases of movable kidney which have been under my care. Mr Morris has operated upon 57 cases of movable kidney without a death. In these 9 were males 48 were females. The right kidney was affected in 45 cases. The left in 12. Of these latter 7 occurred in females 5 in males. No history is given indicative of the cause of mobility, but the chief symptoms are shortly stated, and may be divided according to the classification I have given as follows: Pain the chief symptom in 27. Renal symptoms in 14. Gastro-intestinal in 13. Two of the operations were performed on the same person, both kidneys being affected, so that only one case does not fall under these heads. In it "micturition was very frequent—the right kidney was very mobile and complicated by an abdominal swelling which proved to be a distended gall-bladder." In one case, a left kidney, a mesonephron was found. In this the symptoms were frequent attacks of pain with frequency of micturition, and the urine contained some pus and oxalate of calcium. Mr Morris, in stating his results, claims "that in most the operation has been perfectly satisfactory, and that after nine or ten years he has seen some of the patients whom he has treated by nephropexy with their kidneys as firmly fixed as can be desired, and who have been quite free of their former symptoms since operation."

With regard to treatment of movable kidney, we may consider that three classes of case have to be recognised:—1st. Those in which, as Osler says, no treatment is necessary. The condition is discovered by accident, gives rise to no symptoms, and it is well to say nothing of the existence of the condition to the patient. 2nd. Those that obtain benefit or relief by wearing a pad. Palliative measures succeed short of operation. 3rd. Those in which operation — nephropexy — is necessitated. Let me exemplify this from cases under my own observation.

Mrs V., æt. thirty-one, married, one child. Complaint, appendicitis. Right kidney extremely mobile, but unattended by any symptoms pointing to the kidney, or which can be asso-

ciated with its mobility. At operation for recurrent appendicitis adhesions and a kink of appendix were found. After recovery no pain or other discomfort was complained of by the patient.

Mrs R., æt. forty-six, married, no children ; and Mrs M., æt. thirty-five, married, two children ; suffered from gastro-intestinal symptoms, but chiefly from a dragging pain in the right side and lassitude. In each case, complete relief was got by the use of an air pad fixed to the corset, and applied only after the kidney was carefully noted to be in its normal position. This is, I think, of the greatest importance in treatment. It would be as futile to wear the pad over the kidney when displaced, as to wear a truss with the contents of a hernia in the sac. I believe the air pad fixed to the corset is the most comfortable form of support, and I have found it efficient when a hard solid pad and belt have proved worse than useless. The second patient, Mrs M., had been quite unable to wear the hard pad and belt, but got the greatest comfort from the use of the air pad.

In eight patients on whom I have operated, either because palliative measures had failed upon previous trial, or because the patients refused to try any palliative measure when there was the likelihood of operation proving permanently curative, I find that four, three females and one male, belong to the *simple pain* type. The females were all unmarried, and were respectively twenty, twenty-eight, and thirty-seven years of age. There was nothing indicative of a renal condition in any of them, but in all the kidney (right) was readily palpable and very mobile. In two the kidney frequently occupied the iliac fossa, and in one this, I believe, explained how the condition had been overlooked by several observers. The male patient had a movable right kidney brought about by traumatism—a fall from a horse.

*Three* patients belonged to the *gastro-intestinal* type, and were all aggravated cases. Vomiting of food had led to emaciation, and treatment had proved unavailing to relieve the gastric symptoms. One of these patients was unmarried ; the others, although married, had borne no children.

The last of the eight cases was a married woman of the poorer class who had borne several children. Her left kidney was freely movable, and exhibited intermittent hydronephrosis.

The method of operation I have invariably used is to pass

three deep sutures of strong catgut (Hartmann's) through the kidney substance; using for this purpose MacEwan's herniotomy needle; and stitch the organ to the posterior abdominal wall as high up as possible. I freely separate and remove the perirenal fat, but I do not scarify or strip the capsule proper. In all firm union has resulted.

It may be asked does malposition, although the kidney be fixed, not give rise to symptoms. I have not found it so, and to judge from analogy, we would not expect any inconvenience, as in congenital malposition no untoward symptoms are complained of, and the condition, as a rule, is only discovered after death. A paper dealing with this subject by Dr Newman has been recently published in the Glasgow Hospitals Reports for 1898.

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## Meeting VI.—March 1st, 1899

SIR JOHN BATTY TUKE, *President, in the chair*

### I. ELECTION OF MEMBER

S. W. Carruthers, M.B., C.M., 44 Central Hill, Norwood, London, was elected an Ordinary Member.

### II. EXHIBITION OF PATIENTS

1. *Dr William Elder* exhibited—

(*a*) Case of SKIN ERUPTION for seventeen years following vaccination.

The case was a young man who had had a skin eruption on his right arm which had lasted for seventeen years. It had appeared soon after vaccination and began at the seat of the vaccination mark. It showed all the characteristics of lupus. The patch was oval in shape about 4 inches broad and 5 long.

It had never actually been at any time an open sore, but on several occasions it had been more painful and inflamed-looking than it usually is. It had followed vaccination, the lymph having been obtained from another child, but whether *post hoc* or *propter hoc* it is impossible to say.

(*b*) Case of CONGENITAL ABSENCE OF FIFTH METACARPAL BONE of one hand. The skiagram of this case showed

that the fifth metacarpal bone was completely absent, the fourth and fifth proximal phalanges articulating with the fourth metacarpal bone.

There were also abnormalities in the articulations of the carpal bones.

(*c*) Case of AMYOTROPHIC LATERAL SCLEROSIS, showing in addition to exaggeration of the reflexes and spasm in the muscles of his lower limbs, marked atrophy of the muscles of the lower limbs with tendency to formation of talipes varus in both feet. No sensory affection.

2. *Dr R. Scot Skirving* exhibited a case of CHARCOT'S DISEASE WITH DOUBLE DISLOCATION OF HIP, &c.

Wm. White, 43.

Patient was quite able for his work as sea cook until five years ago, though previous to this he had suffered from 'lightning pains" for several years. He was, in 1894, knocked over by a heavy sea aboard a fishing-smack, and he was removed to hospital, being unable to walk. Since then he has been unable to walk without crutches, which he got on leaving the hospital, where he was for fourteen weeks.

(i) He now presents these symptoms of *Locomotor Ataxy* :—

1. Entire absence of knee jerks.
2. Loss of muscular sense in lower limbs.
3. Argyll Robertson pupil with
4. Contracted right pupil (spinal myosis?).
5. Contracted fields of colour vision with entire absence of green perception.
6. Delayed sensation (especially to pain) in lower limbs.
7. Unilateral facial sweating.

(ii) *Charcot's Disease*

*Painless* dislocation of *both* hip joints, which permits abnormal mobility, allows of the limbs being easily distorted ; the presence of marked "*grating*" sensation upon movement.

(iii) On left thigh, 2 inches below left anterior superior spine is a mass of about 3 inches by 2 inches, fairly freely movable, painless and of hard, almost bony, consistence apparently in the substance of the Tensor Fasciae Femoris.

3. *Dr Cameron and Dr Scot Skirving* exhibited a patient with COMPLETE NECROSIS OF FRONTAL BONE.

This patient, aged 53 years, states that he cannot recollect having had any venereal disease other than "gonorrhœa," at age of 20, for which no treatment was adopted. He married within three years of the "gonorrhœa," and there is no history of his wife having been affected, and he has three grown up children, who show no evidence of inherited syphilis.

The frontal symptoms commenced twenty years ago with pain, actual ulceration dating from twelve (?) years ago.

His present condition is as follows: complete absence of frontal bone, which was easily tilted off *en bloc* a month ago, leaving the pus-covered pulsating dura visible; exposure of the upper surface of the right eyeball, the eyelashes having grown round from beneath the eyelid into the anterior fossa of the cranium; perforation of the palate; and communication of cranial and orbital cavities; corneal opacity and old ulceration of corneæ with loss of vision.

For some years patient had treated himself by flushing the head with a hose attached to a bath-tap, and as the pus could not be got at properly until the bone was removed, the offensiveness of the necrosis was horrible.

As is seen here the necrosed frontal bone is practically entire, with a small perforation on one side.

The chief points of interest are—

Absence of treatment and results.

Marriage within three years and apparent immunity of wife and children.

The size of the necrosis and exposure of contents of right orbit.

(As the Rolandic areas are within the margin of the parietal bone, no motor effect was observed from irritation during dressing or when the dura was syringed.)

4. *Mr H. J. Stiles* exhibited (*a*) an infant, 7 months old, with a LARGE HYDRENCEPHALOCELE. The tumour, which was much larger than the rest of the head, was peculiar, in being slightly sacculated, owing to the presence of four diverticula, of which the anterior extended forward as far as the forehead, the posterior to the sub-occipital region, and the two lateral to the temporal region. The tumour was covered with well developed skin from which an abundant crop of hair had

grown. Transmitted light showed that, with the exception of the basal portion, the sac was translucent; it was not particularly tense, nor did it become more so when the child cried. The patient appeared to be quite blind, but it could evidently hear quite well. The ophthalmoscope showed optic atrophy. The rest of the head was distinctly microcephalic; the eyes were not specially prominent, and the forehead, though narrow, did not recede enough to produce the toad-like appearance seen in an anencephalic fœtus. The child was otherwise fairly strong and healthy, and had recently recovered from an attack of bronchitis. The condition appeared to be a combination of cephalocele and meningocele. Mr Stiles pointed out that the case was of interest in regard to (1) the large and sacculated character of the tumour, (2) the age and the healthy condition of the child.

A point of some interest in the history was, that when the mother was three months pregnant, the abdomen was much more prominent than normal. An experienced gynecologist came to the conclusion that the patient, in addition to being pregnant, was suffering from an ovarian tumour. As soon as the child was born, however, no further trace of tumour could be discovered, so that there can be no doubt, I think, that the "tumour" was represented by the enlarged head of the fœtus, a fact which seems to confirm the view that such conditions originate early in embryonic life.

(*b*) A boy, aged 12 years, showing aggravated crippling, the result of EXTENSIVE ACUTE ANTERIOR POLYOMYELITIS. The attack occurred when he was about a year old. He went to bed apparently quite well, but on the following morning "his whole body appeared to be paralysed," and "for a week he was unable to move." The power gradually returned in the left arm, and to a considerable extent also in the right arm. There is now complete paralysis and great wasting of all the muscles below the knee. The feet occupy the paralytic equino-varus position; the knees are acutely flexed, and cannot be extended. The thighs are also greatly atrophied; the quadriceps is completely paralysed, the flexors and adductors only partially. The patient is with difficulty able to shuffle along on his knees, the psoas and glutei being the muscles most concerned in the progression. The legs are dragged helplessly after him, and specially constructed boots are worn upon the

knees. As regards surgical interference, Mr Stiles was of opinion that the legs constituted such an encumbrance that the patient would be much better without them; he proposed, therefore, to amputate both below the knee, at the seat of election. The bones and the muscles of the thigh, as well as those of the leg, were so atrophied that it was not considered advisable to substitute ankylosis of the ankle and knee joint for amputation.

(*c*) A child, aged three and a half years, with double coxa vara. When first seen, six months ago, the patient was markedly rachitic, and was scarcely able to stand without support. The femora were arched forwards and outwards, and both lower extremities were rotated outwards as far as the coronal plane. By manipulation the left leg could be rotated outwards to such an extent that the foot could be made to point directly backwards. The lumbar spine was arched forward; the trochanter here displaced upwards and backwards, so that Bryant's ileo-femoral triangle was modified in the manner described by Ogston. The child, who had now been under anti-rachitic treatment for six months, could walk fairly well; the eversion was much less marked, but there was still a tendency to knock the heels together in walking.

Mr Stiles was of opinion that if the improvement continued osteotomy, with the view of counteracting the eversion, would not be called for.

## III. Exhibition of Specimens

1. *Mr H. J. Stiles* exhibited—

(*a*) The ABDOMINAL VISCERA from a boy aged five years who had suffered from general lymphosarcoma of the abdomen. The whole peritoneum was greatly thickened. The mesentery varied from half to one inch in thickness. The diaphragm was infiltrated with nodules and plate-like areas of lymphosarcoma. The liver contained many isolated tumours varying in size from a small pea to a walnut; these appeared to be spreading throughout the liver from the portal fissure. The gastro-hepatic omentum was also greatly thickened; while the great omentum, although less thickened, had a peculiar arborescent appearance, due to the small-celled tumour formation being distributed along the course of the vessels. The right kidney was normal; the pelvis of the left was considerably dilated, and a small,

secondary deposit was found immediately under the capsule. The wall of the left ureter was greatly thickened and almost obliterated; the right ureter was in much the same condition, though less marked. In the neighbourhood of the hilus of the spleen was a sarcomatous mass, which was evidently spreading into the substance of the organ along the course of the vessel. The pancreas and supra-renals were normal. The aortic and iliac lymphatic glands were only slightly enlarged. The disease appeared to have originated in the lymphoid tissue of the intestine and mesentery. In spite of so much disease, the boy had been running about apparently quite well ten days before he was admitted to the Children's Hospital.

He complained of swelling of the abdomen, diarrhœa and cramp-like pains. He was thought to be suffering from tubercular peritonitis. Operation was decided upon. After opening the abdomen the appearances presented did not suggest tubercle, and the probability of lymphosarcoma was considered. With the exception of the removal of a small portion of the omentum for microscopic examination, the parts were not subjected to further interference; the child, however, suffered greatly from shock, and died twelve hours after the operation.

(*b*) The THORACIC VISCERA from a child who suffered from severe dyspnœa with expiratory stridor.

The preparation is from an infant, aged 4 months, who died eighteen hours after tracheotomy. The operation was performed on account of severe dyspnœa, accompanied by an expiratory stridor and all the signs of obstruction to the entrance of air into the lungs. The dyspnœa had existed for a month, and had become more marked during the last week. The child was in a critical condition when seen by Dr John Thomson, who at once sent it to the Sick Children's Hospital. The throat was normal. The history, and the fact that the stridor was expiratory, pointed to the trachea as being the seat of obstruction. It was deemed advisable, however, to give the child the benefit of any doubt, and to perform tracheotomy. The breathing was in no way improved by the introduction of the tube.

The specimen shows that the lower end of the trachea and origin of the left bronchus were much flattened by the pressure upon them of a firm tuberculous gland the size of a small walnut. Mr Stiles had recently met with two other cases of

severe respiratory obstruction in infants, due to the pressure of tubercular glands upon the lower end of the trachea; in both instances the stridor was *expiratory*, and not inspiratory, as in the case when the obstruction is laryngeal. He (Mr Stiles) regarded the expiratory stridor as due to the additional pressure, which was caused, during expiration, by the weight of the chest wall. Should further experience prove this to be a pathognomonic symptom of tracheal obstruction in children, it affords us a means of discerning an important form of respiratory obstruction in which the operation of tracheotomy would be futile.

*Dr Scot Skirving* exhibited—

(*a*) NECROSIS OF FRONTAL BONE. Entire sequestrum from syphilitic patient exhibited at same meeting.

(*b*) TUBERCULOSIS OF MAMMA. Breast from girl of 19 years, removed by Dr P. H. Maclaren. Externally a sinus had existed for many months, opening on to skin. Internally numerous typical tubercular foci scattered throughout breast. The largest of these was the size of a cherry. Microscopically, a few perfectly typical giant cell systems were found. These were in the connective tissue and not in the gland substance. No bacilli could be demonstrated.

(*c*) COMPOUND FRACTURE OF RIGHT CARPUS. Dissection of hand and wrist from arm amputated by Dr Scot Skirving. The specimen shewed fracture of os magnum and semilunar bones with chipping off of portions of both styloids. In addition, as the result of the great violence of the accident—a fall from a second story on to the palms—fracture of the radius about its middle.

(*d*) MICROSCOPES illustrating tuberculosis of mamma.

## IV. EXHIBITION OF INSTRUMENTS

*Mr C. W. Cathcart* exhibited what he described as a "CLINICAL RESEARCH CASE." This consisted of a small wooden box containing all that was necessary for staining deposits or discharges for micro-organisms, or for cutting, staining, and mounting fresh sections of tumours. His object was to facilitate these modes of investigation by the students, and he had found it very useful. A senior student was de-

tailed off as "pathological" clerk, and was entrusted not only with the charge of the pathological specimens, and with the preparation of slides for the microscope, but also with the duty of demonstrating them to the other students. He had had some difficulty in planning the box to make it both simple and efficient, and he was indebted to Dr T. Shennan for many useful suggestions in detail. The "case" was made by Mr Fraser in Teviot Place, and cost (including the improved microtome) about 50s.

## V. Original Communications

### 1. PERITONITIS AS A CAUSE OF INCREASED PERISTALSIS IN THE RECTUM AND IN OTHER PARTS OF THE INTESTINE

By Charles W. Cathcart, F.R.C.S. Engd. & Ed., Senior Assistant Surgeon, Royal Infirmary, Edinburgh

We are all so familiar with paralysis of the bowel in acute peritonitis, that we are apt to think that increased action of the bowel can hardly be due to the same cause acting under different circumstances. I for one did not realise it till the following case brought it home to me.

#### Case I.—*Pelvic Peritonitis*

About five years ago Dr P. A. Young asked me to see with him, late one night, a married woman, aged 37, who showed certain obscure abdominal symptoms, and was in an anxious condition.

*History of Attack.*—About midday, while sitting in a tramway car, she had been suddenly seized with intense abdominal pain, and had only with the greatest difficulty dragged herself from where the car left her to the rooms in which she was living. Dr Young saw her about half-an-hour after she got home, and found her lying on the top of her bed half undressed, faint and collapsed, and with a bed-pan under her. Her bowels were being so frequently moved that she thought she had "dysentery." There was nothing in the bed-pan, however, except urine and a little mucus from the bowel. She felt cold and shivery, pulse 100, temperature normal. She had vomited

several times, and complained of great pain, felt chiefly about the umbilicus, which came on in severe paroxysms, with intervals of about two minutes. The abdomen was tender to touch. Various palliative measures were adopted, and when I saw her the pain was less, and the tenesmus had diminished. She had an anxious look, and was very nervous about herself. Pulse was then about 108, temperature normal. The abdomen on inspection was not distended : respiratory movements impaired. The muscular wall was resistant all over, especially in the left iliac fossa, and there was a good deal of tenderness over the lower part of the abdominal wall. *Per rectum* there seemed to be a little "ballooning," but nothing definitely abnormal. The walls of the vagina seemed unduly hot, but that was all.

*Previous History.*—She had been married for twenty years, and had two sons aged respectively 18 and 19. Shortly after the birth of the second son she had had a miscarriage, but it did not seem to have left any ill effects, for her menstruation had gone on regularly at three weeks' intervals. It had usually lasted two or three days, and had caused no trouble. She had thought her heart weak on account of shortness of breath on going upstairs, but Dr Young had been unable to make out anything to account for this. She was on a visit to Edinburgh. A few days before this sudden seizure she had been in bed with a sharp attack of follicular tonsillitis during the progress of a menstrual period. Although recovering from this attack she had not felt well ; her tongue had been furred and her liver and stomach had been apparently out of order. On the morning on which she was taken ill her bowels had been moved several times with a powder (rhubarb and grey powder) which Dr Young had prescribed.

*Diagnosis.*—We found it very difficult to make up our minds about the case, but thought that fæcal impaction in the sigmoid flexure was a feasible and the most probable diagnosis. There was no reason for thinking that the pelvic organs had been diseased ; and the region of the vermiform appendix did not seem involved. On the other hand, colic from fæcal impaction sometimes causes great collapse, and although she said that her bowels had always been regular, and that they had moved loosely on the morning of the seizure, we could not on that account exclude the possible presence of fæcal masses. In the rectum at least they may set up irritation, and cause a loose

catarrhal discharge which finds its way past them. There were certainly symptoms pointing to peritonitis, but in the absence of anything to explain its occurrence, and under the belief that colic might simulate such symptoms, we thought it better to treat the case by palliative measures, and watch its further development.

*After-history.*—During the night the patient sent for Dr Young on account of a feeling of irritation in the bladder. This was relieved after the use of the catheter, and next morning she did not complain of it. Her general condition in the morning seemed much the same, but her pulse was quicker—that is, 115, and her abdomen was more swollen and tender. Throughout the day she grew weaker, and about 5.30 P.M. took a sudden turn for the worse, became cold and collapsed, and died at 6.30 P.M., about thirty hours after the seizure in the tramcar.

*Necropsy.*—At the *post-mortem* examination we found the pelvis full of grey grumous fluid, and recent lymph glued together all the coils of intestine there. The ovaries and tubes were healthy, except for the signs of recent inflammation ; both tubes, however, contained fluid exactly like that in the pelvis. Mr Stiles kindly examined for me the fluid from the tubes and that taken from the pelvis, and reported the presence of streptococcus in both. There were no fæcal masses in the large intestine.

This was therefore a case of very severe pelvic peritonitis from the streptococcus. The etiology of the disease is very interesting. I do not wish to divert attention from the main purpose of this paper, but in passing let me say that I believe streptococci were carried by the circulation from the throat to the Fallopian tube or tubes ; that an accumulation of fermenting fluid formed there, and that its sudden escape into the pelvis was the cause of the attack in the tramcar which so soon ended in death. The marked tenesmus for the first few hours of the illness led us astray, by making us think of the inside rather than of the outside of the bowel as the seat of the lesion.

About a year after this Dr Young asked me to see another case with him, which, although differing from the last one in some respects, resembles it in others, and has an important bearing on our present subject.

## Case II.—*Gangrenous Perirectal Abscess*

*Condition of Patient when first seen.*—A young married woman, about five months pregnant with her first child, had sent for Dr Young hurriedly on account of symptoms which at first sight had seemed to be those of impending miscarriage. She was suffering from very severe bearing-down spasms, which he found, however, were affecting the rectum and not the uterus. When I saw her late at night the spasms were still going on at intervals of a few minutes. During the spasm she held her breath, and her whole body was strained as in a uterine contraction; the perineum was forced down, and the mucous membrane of the rectum was violently everted. Nothing, however, came by the bowel except a little mucus. Inspection between the spasms shewed that the perineum remained swollen, especially on the right side, but the skin over the swelling was not discoloured; on percussion this swelling was distinctly resonant, and it had a soft fluctuating feeling on palpation. When examined from the vagina and rectum, the interior of the pelvis, had a soft and boggy feeling.

*History of the Attack.*—She stated that about a fortnight before I saw her she had been overheated and tired in hurrying to hear a public lecture, and had been chilled while listening to it. Next day she complained of pains in her back and legs, which steadily grew worse, and were aggravated by her going to nurse a friend in a miscarriage a few days later. Afterwards the pain had been felt in the neighbourhood of the anus and down the hips, and, as she had a tendency to piles, Dr Young had recommended hot fomentations and a sitz bath. She got no relief, however, and micturition became increasingly difficult, until three days before the tenesmus began the catheter had become necessary, and had remained so. On the morning of the day on which I saw her she was straining at stool, as the result of having taken some opening medicine, as she thought. Suddenly something seemed to give way about the back passage; the lump appeared in the perineum, and the tenesmus and bearing down began. There had been no vomiting, and except during the spasms there was no abdominal pain.

*Diagnosis.*—The sudden appearance of the perineal swelling, its marked resonance, and the absence of redness of the

skin over it seemed to point to hernia forcing its way through the pelvic floor. The boggy feeling of the interior of the pelvis I thought might also be explained in this way. If a hernia, it was evidently not strangulated, so, as my efforts to reduce it under chloroform were unavailing, she was left over the night with a bandage on the swelling and the pelvis raised. I hoped that gravitation and pressure might do gradually what manipulation had failed to effect.

*After-history: Recovery.*—By next morning her urgent symptoms had disappeared. During the night a large collection of fœtid fluid and gas had burst through the vagina, and she had felt instant relief. The perineum had to be freely incised, and extensive sloughs were removed. The abscess had completely surrounded the rectum as far as the finger could reach. Dr Young attended her through a miscarriage which occurred within a week, and by vigilant care and watchfulness prevented any contamination of the uterus from the abscess, which was discharging in the perineum and through the vagina. She is now in excellent health, with the affected parts soundly healed.

This was a case of gangrenous perirectal abscess. The extreme tenesmus must have been due, as in the first case, to irritation round the outside of the rectum, only it was in the cellular tissue instead of on the surface of the peritoneum.

Having been forced by these two cases to recognise that increased action of the rectum may sometimes be associated with inflammatory conditions outside and not inside the bowel, I was on the look-out for further illustrations of this symptom. The condition of the anus in perirectal cellulitis secondary to acute prostatic inflammation seemed to be one. Not only is there increased frequency of micturition, and straining at stool in such cases, but the anal sphincter seems unable to regain its tone. The anus becomes quite patulous and relaxed and allows the finger to be inserted without resistance. A condition like this has also been observed in advanced cases of senile enlargement of the prostate, but in the light of the cases just narrated the explanation usually offered for that condition does not seem adequate. It is said that the very frequent straining to empty the bladder, during which the anal sphincter is also relaxed, lead to a continuous relaxation of the latter. This may be partly true, but does it not seem also probable that irritation

from the wall of the rectum acting reflexly may be another factor in the condition, especially since in many of such cases inflammation in and round the prostate is superadded to the senile enlargement? On the other hand, frequent micturition, due to irritation of the vesical mucous membrane alone is not accompanied with anal relaxation, or if at all, to a much less extent.

These considerations were present in my mind when I was asked to deal with the following case last summer:

### Case III.—*Pelvic Abscess*

*History.*—It was that of a student in the students' ward of the Edinburgh Royal Infirmary. Mr Caird had opened a fœtid appendicular abscess in the right side about ten days before, and being out of town for a day or two had asked me to take charge of the case in his absence. For about ten days after the operation progress had been satisfactory, but for a few days latterly the patient had not been so well. He had been duller, less inclined for food, pulse somewhat more rapid, and temperature inclined to rise. On the morning when I saw him a decided change for the worse had set in. His face was flushed, and his conjunctivæ had a tinge of yellow; he was very drowsy and stupid. His temperature and pulse, if I remember rightly, were about 100. His abdomen was distended and tympanitic, but not tender. The nurse casually mentioned that he had had some diarrhœa, gradually increasing for the last day or two, and that he did not seem able to control the evacuations. She thought it due to increasing weakness.

*Diagnosis.*—On examination I found the anus patulous and a boggy feeling in the pouch of Douglas, which on bimanual examination amounted to indistinct fluctuation. Here, then, the symptoms seemed those of septic poisoning rather than of peritonitis; and the diarrhœa, patulous anus, and result of anal examination made it highly probable that an accumulation of fœtid pus had occurred in the pelvis. Possibly an examination without the rectal symptoms ought to have been sufficient. They might, however, have been interpreted differently, and it was a great comfort to feel in urging an immediate operation that they strengthened the view one took instead of weakening it.

*Operation.*—He was put under chloroform without delay. I worked down in the middle line just above the bladder, and after separating coils of adherent intestine, I had the pleasure of reaching in the pelvis a small collection of most offensive pus. After this the patient made an uninterrupted recovery.

On referring to some of our text-books I find sufficient notice of such symptoms to confirm these observations, but a sufficient absence also of notice to justify this attempt to bring them more prominently into notice. Thus as regards pelvic perirectal abscess, there is no allusion to tenesmus as a symptom in Allingham's, Ball's, Cripps's, or Matthew and Smith's works on the rectum. In some of the older books, however, there is. Thus Percival Pott (1) alludes clearly to the straining and tenesmus which accompany such abscesses, and so does Ashton (2).

In the account of pelvic peritonitis, moreover, no mention is made of accompanying rectal symptoms in Zweifel's *Cyclopædia*, nor in Hart and Barbour's *Manual*. In Pozzi's work on *Gynæcology* (translated by Wells, 1892), however, there is a passing reference to this symptom, but it is mentioned along with others, and no especial importance is attached to it.

But if peripheral irritation is able to excite the action of the rectum and simulate diarrhœa or dysentery, we may naturally ask if a similar phenomenon may not be found in connection with other parts of the intestine. On examination we find that it is so. In the first place, it has been definitely proved by experiment. In 1896 A. S. Grünbaum read a paper at the meeting of the British Association on the effect of peritonitis on peristalsis. Only extracts of the paper have been as yet published (3), but Dr Grünbaum writes, in answer to my inquiry, that "The gist of the whole point investigated can be put in a few words. In rabbits the introduction of irritants, whether chemical like turpentine, or bacterial like cultures of bacillus coli communis, cause increased peristalsis for about twenty-four hours. All portions were not equally affected, but the rectum was very decidedly. The inquiry was undertaken at Professor Nothnagel's request at Vienna." In the abstract which appeared in the *British Medical Journal*, it was said that:

"In the first twenty-four hours the peristalsis of both large and small intestine was increased; it then gradually diminished

in about four days. The large intestine became paralysed before the small."

*Apropos* of this inquiry, Professor Nothnagel (4) says :

" We have just been speaking of paralysis of the bowel in acute diffuse peritonitis. It would be a mistake to describe that as a necessary symptom. The relations of peristalsis are rather the following. Numerous clinical observations show that in acute diffuse peritonitis normal evacuation, that is, a normal degree of peristalsis, is found in individual cases. In puerperal peritonitis, peristalsis may be even increased. To this one might fairly reply that the cause of diarrhœa was not the anatomical process of inflammation as such, but other causes acting indirectly. However, clinical observation and the results of experiments upon animals which Grünbaum undertook at my suggestion, teach us that in other forms of peritonitis also a distinct increase of peristalsis may be present at the beginning. Hence paralysis of the bowel can in no way be described as an invariable symptom of acute diffuse peritonitis."

Such clinical observations as Professor Nothnagel refers to may be found in Treve's well-known lectures on peritonitis (5). He says :

" In 100 cases of peritonitis from the London Hospital the bowels were classed as " loose " in 28 instances. In some of these 28 cases there was actual diarrhœa, in other the bowels acted with frequency and without artificial aid ; while in a third series of cases a free action was maintained by enemata or by aperients. In two cases of peritonitis in which diarrhœa was met with, it was noteworthy that the peritonitis did not appear until after the diarrhœa had set in, a circumstance which suggests the action of the colon bacillus."

By this he seems to indicate that the diarrhœa might have led to the diffusion of the colon bacillus, and thus might have caused the peritonitis, but Grünbaum's experiments make it quite possible to suppose that the peritonitis caused the diarrhœa.

But there seems to be still another way in which experiment and clinical observation mutually throw light on one another. In the Bradshaw Lecture for 1892 upon the Signs of Acute Peritoneal Disease, Dr Gee makes the very interesting suggestion that the pain of early acute peritonitis is really due in great measure to colic. Thus, after having pointed out how

difficult it sometimes is to distinguish colic from peritonitis, he continues :

" But it may be that in contrasting thus the pain of peritonitis with the pain of colic, we are seeking for a distinction where there is no real difference. Indeed, it is highly probable that the earlier physicians were often right, and that much of the spontaneous and paroxysmal pain of peritonitis is due to colic, that is to say, to vermicular contractions of the intestines much more powerful than usual. Surgeons tell us that when they open the abdomen in order to remove an ovary, the intestines are seen to be at first in very active movement ; and that these movements may be painful is proved by what we sometimes observe in intestinal tympanites, when the peristaltic contractions of the bowels can be seen through the abdominal walls, each contraction being attended by an unnatural sensation or by actual pain."

From some remarks in other parts of this lecture, it would appear that Dr Gee did not consider that peritonitis might cause an increase of ordinary peristalsis. Had he done so, however, it would only have increased the probability that his suggestion as to colic was correct.

We are now, I think, justified in believing that as the result of irritation round the intestine (produced most frequently by peritonitis), the following symptoms may be produced, that is, increased peristalsis amounting sometimes to severe spasm in the small and greater portion of the large intestine, and relaxation of the sphincter, frequent evacuations, and, in severe cases, intense straining and tenesmus, when the walls of the rectum are involved.

It is not, however, necessary to suppose that such symptoms will be prominent or even recognisable in every case of peritonitis or of perirectal irritation. If we take into account the following considerations this will not be difficult to understand. The peristalsis of the small intestine may be so irregular or so transient that it may not show itself as diarrhœa. Grünbaum found that the large intestine became paralysed before the small ; hence the effect of the increased peristalsis might only be to fill the large intestine with an accumulation of fæces and gas. Again, in relation to the rectum it might be that certain forms of organism which often produce pelvic abscesses such as the gonococcus do not

cause an irritation which is sufficiently intense to give rise to the symptoms in question. Or, again, it may be necessary that a certain area of the rectal wall should be affected in order that a perceptible result should be produced, and pre-existing adhesions may prevent this.

But, on the other hand, it seems not unreasonable to extend our view of the possibilities of reflex effects upon the intestine. If the afferent nerves of the intestine (whether of the mucous or peritoneal surfaces) can reflexly stimulate the intestinal muscular coat, why not afferent nerves belonging to the same splanchnic system but coming from other viscera in the abdomen, pelvis, or even thorax? This would explain the presence of colic from renal or biliary calculus, or from a severe onset of pneumonia, as in such cases as Mr Symonds has recently referred to (6). Already we know that the walls of the stomach are reflexly affected by such irritation, for vomiting is a common symptom in such cases.

Although we have confined our attention to reflex excitement of the intestinal muscular coat from irritation, we must not forget the possibility of a reflex paralysis coming on at a later stage, and thus increasing the paralysis due to involvement of the muscular coat itself.

In conclusion we may briefly consider some of the advantages to be derived from the knowledge gained from this study of a few symptoms:

1. Such cases as the first two which I have just described will be clearly understood, and therefore promptly and efficiently treated when they present themselves.

2. The possibility that increased action of other parts of the intestine may be due to a cause outside and not inside the walls of the bowel may often help us to account for otherwise obscure symptoms in ways perhaps that we cannot as yet realise.

3. The probability that colic may in like manner be caused by irritation outside as well as inside the bowel may be most valuable in helping us to form a diagnosis in a difficult case. Hitherto most of us have only recognised that many of the symptoms of peritonitis and of colic (intestinal, renal, or biliary) are often very much alike. Now we may be prepared to believe that some of them are identical. This, instead of discouraging us, should have the opposite effect. We shall know what symptoms to accept as possibly common to several

causes, and we shall not put too much stress upon them. This will make us seek more eagerly for points in differential diagnosis. Thus perhaps symptoms which otherwise might have been missed or passed over as unimportant will be found lying ready to our hand as invaluable guides when we understand how to look for and use them.

References.

(1) "On Fistula in Ano," second edition, p. 22.
(2) "On the Rectum and Anus," p. 221.
(3) *British Medical Journal*, vol. ii., 1897, p. 1159.
(4) "Die Erkrankung des Darms und des Peritoneum," p. 571.
(5) *British Medical Journal*, vol. i., 1894, p. 456.
(6) *British Medical Journal*, vol. i., 1899, p. 517.

## 2. THE ETIOLOGY OF GOUT

### Observations on the Pathological Relationships of Uric Acid

By Chalmers Watson, M.B., M.R.C.P.E.

In the wide domain of medicine there are few, if any, diseases which figure more prominently in every-day medical work than that of gout. Apart from its frequency, there are other factors which tend to make a study of it, one of more than usual interest. Of these the more important are found in relation to the etiology and treatment. In connection with the former we have to note that while the disease has been recognised from time immemorial, and has been the subject of much careful investigation and still more speculation, our knowledge of its cause is still very imperfect. We certainly possess a much more accurate knowledge of certain details bearing on the chemistry of uric acid, but we can hardly say that our ideas as to what gout really is are much better defined than those of the older writers. In connection with the latter factor—the treatment—the markedly diverse views entertained as to the best method of treatment by diet, and various medicinal means, speak still more eloquently of our ignorance of the subject.

In the first place we must seek to define what we mean by the term gout. This is of special importance, in view of the great frequency with which a diagnosis of the condition is made. For my own part I have seen no definition which succeeds in fully satisfying on the one hand the scientific ideas of the pathologist, and on the other the more practical require-

ments of the practitioner. Let us look for a moment at some of the definitions commonly employed. Thus we find it regarded as "a condition characterised by the deposit of urate of soda in the joints or tissues of the body." Such a definition may be true from a strictly pathological point of view, but it is too narrow to appeal to the practical physician. In this connection it is of some interest and importance to refer to a paper by Dr Norman Moore on the Morbid Anatomy of Gout (St Barth. Hosp. Reports, 1887). He, as a pathologist, considered that gout existed wherever free urate of soda could be discovered in any cartilage of the body, and his investigation, which was a very detailed one, comprised the *post-mortem* examination of over 70 cases. In some of the cases recorded no history of attacks of gout had been obtained from the patient during life.

Two of his conclusions may be quoted :—

1. It is common to find urate of soda in the joints of those persons whose aortic valves show chronic degenerative changes with calcification. (The application of this point to many of our hospital cases is at once apparent, and perhaps not sufficiently recognised.)

2. Urate of soda is present in the joints of a large proportion of those persons over 40 years of age who die of cerebral hæmorrhage.

A second wide-spread view is that which characterises gout as "an enduring tendency to the accumulation of urates in the bodily fluids and to their formation as crystals of sodium biurate in the tissues." This description, though at first sight one which rather commends itself, will not stand careful consideration. Full evidence in support of this statement will be given later, but one point may here be referred to. In leucocythæmia we have a condition where the blood is impregnated with uric acid to an extent, and for an enduring period, unknown even in the most aggravated cases of gout, yet in this disease we find clinically no manifestations of gout, and pathologically no evidence of uratosis.

Again, Dr George Balfour, in a paper read before the Society last year, spoke of gout "as a meaningless epithet vaguely applied by physicians to all ailments occurring after middle life which are obscure in their origin, variable in their symptoms, and not readily amenable to treatment." This description is a fairly true one, and indeed I think it could be amplified to

include many ailments encountered before middle life, but at the same time it is a description possessed of more clinical interest than scientific value.

All physicians are agreed as to the diagnosis in well-marked cases of acute gout, and in such cases the facts of hereditary history are quite immaterial. All, further, are at one as to the diagnosis in cases of chronic gout, characterised by typical gouty enlargement of the smaller joints, some aberration in the excretion of uric acid, and associated functional or organic derangement of the digestive, cutaneous, nervous, or other systems. But these two classes of cases form a very small proportion of those frequently diagnosed as gout or goutiness. I can best amplify this point by a few practical illustrations drawn from ailments encountered at different ages and commonly considered gouty in their origin. Thus it is not uncommon to find infants, of gouty stock, passing large quantities of uric acid crystals, their passage being associated with restlessness, screaming and general systemic disturbance. A little later we not infrequently find children, also of gouty stock, affected with various cutaneous eruptions usually associated with a sensitive intestinal mucous membrane, these symptoms recurring with such frequency as to convince the practitioner of the existence of a causal relationship between them and the parent's disorder. Still later we see a young woman aet. 20 complaining of dyspeptic symptoms, slight or marked mental depression, with occasional copious sediment of uric acid in the urine. Yet another type of case might be quoted, a young man of thirty with marked hereditary tendency to the disease, after returning from his autumn holiday, spent mostly on the moors, settles down to office work, and in less than a week complains of "Lumbago." (He considers his back has always been weak since a severe muscular sprain.) A cursory examination suffices to show the case to be one of urolithiasis—the seat of the pain being the pelvis of the kidney—the cause of it uric acid. The interest of such a case from the point of view of a study of metabolism and conditions which modify it, is at once apparent. At and after middle life it is still more easy to furnish instances of disease which at any rate appear to be illustrations of that error of metabolism which we call gout, but it is unnecessary to expand on this point. I must pass more directly to speak of the *materies morbi* of the disease, uric acid.

To speak of gout is synonymous with thinking of uric acid. As the late Sir Wm. Roberts said, "An abnormality in the destiny and disposal of uric acid is a fundamental element in any scientific conception of the gouty state and a dominant factor in the genesis of its chief symptoms and physical signs." Here we have, if not a definition, at least a description which is alike admirable in its conception and guarded in its tone, and there is great need for caution as to statements made about so complex a subject. Uric acid produces morbid symptoms in three ways—

1. In connection with acute gout.
2. „ „ chronic gout.
3. „ „ urolithiasis, that is the deposit of free uric acid in the pelvis of the kidney, in a lower part of the urinary tract, or in the vessel after voiding. In this connection one must bear in mind that the mere deposit of uric acid crystals even in considerable amount does not necessarily indicate the passage of excess of uric acid. The two conditions—gout and urolithiasis—are included in the term gouty diathesis, which may be characterised by, on the one hand,

1. A tendency to the accumulation of uric acid in the blood in the form of a salt—urate of soda—a condition termed by Roberts uratæmia.

2. A tendency to the deposit of urates in the tissue, a condition termed uratosis.

This brings me to speak for a moment about uric acid. This salt has, stated shortly, the chemical formula $\overline{H_2U}$, and forms three classes of salts :—

(1) Neutral Urates—$Na_2\overline{U}$—when the metal takes the place of all the displaceable H. This salt cannot exist in the body and is therefore of no present interest.

(2) The Biurate—NaHU—the metal takes the place of half the displaceable H.

(3) The Quadriurate—($NaH\overline{U}\ H_2\overline{U}$)—when the metal takes the place of $\frac{1}{4}$ of the displaceable H. of two molecules of $\overline{H_2U}$.

Those two latter salts are the ones that concern us. The former does not occur naturally in the body. It is a very insoluble salt and when present in the body tends to be deposited in the tissues. The latter is the form in which uric acid normally appears in the urine. It is an unstable body, and may after a time unite with the sodium carbonate of the blood to form sodium biurate, and if this body be produced in larger amount than the

fluids and tissues can hold in solution it becomes deposited in certain tissues. If we review the two conditions previously mentioned in the light of these observations we see that in *urolithiasis* the defect lies in the tendency to a breaking up of the unstable quadriurate and a setting free of its $H_2\overline{U}$—thereby producing gravel and calculus—whereas in *gout proper* the defect lies in the converse direction—the tendency being towards the transformation of the quadriurate into the more stable biurate and the precipitation of this latter in the tissues. Further, it is well again to emphasise the distinguishing features of uratæmia and uratosis. In the former the urates circulate in solution and are therefore incapable of acting as a mechanical irritant. Any injurious action exerted by them must be due to their acting as a chemical poison, whereas in uratosis the urates are deposited in solid form in the tissues and are therefore capable of acting as a mechanical irritant. Further, whereas it is highly probable that uratosis cannot occur without pre-existing uratæmia, it is well known that uratæmia may exist without inducing evidence of gout. The most striking illustration of this has already been referred to, leucocythæmia. We find further illustrations in Bright's disease, various anæmias, and certain other conditions.

What then is the relationship between uric acid and gout? A causal relationship is almost universally admitted, but beyond this, dissentient views prevail. These various views must be briefly summarised.

1. We find the uric acid compound regarded as acting passively and physically while in the crystalline state. This view is supported by Sir A. Garrod and the late Sir W. Roberts, who regard gout, in so far as its phenomena depend on uric acid, as a disease the manifestations of which are proximately due to mechanical injury. Sir W. Roberts, especially considered that uric acid produces no harmful results so long as it remains in solution, the mischief resulting from it (either in acute or chronic gout) depending on its precipitation as sodium biurate in the tissues or fluids of the body.

2. We find the uric acid compound regarded as acting as a poison or irritant while in the dissolved state. The writers who support this view, and it has much to commend it, consider that apart from the local trouble in joints caused by the deposited sodium biurate acting as an irritant, the soluble uric acid compound circulating in the fluids of the body acts as a poison, the

toxic effects of which are responsible for a number of symptoms associated with the gouty state. The supporters of this view, however, fail to take account of one point which seems to me of the first importance and to which I will refer later.

3. A third view is that which considers morbid changes in the structure of tissues as the primary cause of gout. Ebstein is the great exponent of this view. He holds that uratic crystals are only deposited in necrotic tissues, and that a destructive process always precedes the deposition of uric acid, both processes, however, being due to uric acid though in different states of combination. Dr Ord, on the other hand, while regarding degenerative changes in the tissues as the primary cause of gout, does not consider that the uric acid plays an important part in their production.

4. A further view is that which regards nervous disturbance as the primary cause of gout. Sir Dyce Duckworth is the main exponent of this theory, which regards gout as a functional disorder of a definite tract of the nervous system.

These views thus agree in ascribing to uric acid an important rôle in the etiology of gout. But they do not strike at the root of the matter, and this brings me to the threshold of that curious and interesting condition, the uric acid diathesis. In order thoroughly to understand this diathesis it is essential that we have an accurate knowledge of the genesis and physiological significance of uric acid. It is evident that a satisfactory knowledge of the deviations in the production and excretion of uric acid encountered in disease, cannot be obtained so long as we have only an imperfect knowledge of the relation of that substance to the healthy organism. It is the absence of such accurate knowledge that is mainly responsible for many of the diametrically opposed views entertained as to the etiology of gout. As Ewart, in his recent admirable work on "Gout and Goutiness," writes: "Far from being explained, the problem of gout seems to have grown with each successive effort to solve it. New theories are less wanted than a weeding out of some of the older ones. We should build upon well ascertained facts only, and be content at first to ascertain, if possible, what gout is not, leaving it for the future to show what gout is."

This brings me to my own investigations of the subject. After full consideration of the views advanced by recent autho-

ritative writers, I found that the first step necessary was the determination of certain points in the physiology of uric acid. And first let me briefly indicate one or two elementary points that are essential to a clear understanding of what is to follow. The element nitrogen is second only to oxygen in regard to its importance in human life. It is one of the most important elementary constituents of all cells, and it is at the same time a necessary ingredient of food. As a result of nitrogenous metabolism in man and mammals the main bye-product is urea, whereas in birds and reptiles it is uric acid ; but in the mammal a small quantity of uric acid is also excreted, the daily amount in man being half a gramme. If we bear in mind that it is by no means easy to prepare urea from the blood of mammals, even although the amount of that substance excreted daily is about sixty times greater than that of uric acid, it is not surprising that great difficulty has been experienced in recognising the presence of the latter in the blood of a mammal. For a fuller investigation of the subject we must have recourse to examination of the blood and viscera of birds and reptiles, whose main nitrogenous bye-product is uric acid. Here again we must bear in mind that even although uric acid be present in relatively large amount in the urinary excretions it would not be surprising if the amount present in the blood at a single examination were very small. In a recent paper in the *British Medical Journal*, I entered with some detail into the question of the various theories held as to the seat of production of uric acid. It will suffice to say here that the following three views are supported.

1. That the liver is the main seat of its formation.
2. That the kidney is the main seat of its formation.
3. That the spleen and other nuclein holding tissues are the main seat of its formation.

The second view, that which regards the kidney as the main if not the only seat of formation of uric acid in health, has as its strongest supporters in this country, Sir Alfred Garrod and Dr Luff. From the unique position which Sir A. Garrod holds as an authority on the clinical features and pathology of this condition, statements and theories advanced by him are vested with more than usual interest and importance. As my observations have led me to conclusions which are at variance with his most recent teaching on the subject, I must briefly

refer to that teaching before stating the grounds on which I venture to differ from it. In a paper on the "Blood of Birds" in the Proceedings of the Royal Society, published in 1893, he writes: "The blood of healthy birds, instead of containing uric acid as would naturally be supposed, is usually quite free from that substance, except when it has been introduced into the system through the stomach or by injection"; and further "The existence of uric acid in the blood may be looked upon therefore as a morbid phenomenon"; and in summing up from his observations on their application to the human subject, writes as follows: "When uric acid is not introduced into the blood by the alimentary canal, its presence must, according to my view, be accounted for by its absorption into the blood from the kidneys after its formation in these organs, and the salt is necessarily changed from the urate of ammonium to the biurate of sodium."

Dr Luff, writing on the same subject, says: "I considered that these observations of Sir A. Garrod as to the absence of uric acid from the blood of birds were of so great importance, in view of the opinions which I strongly entertain that uric acid in health is only formed in the kidneys, that I thought it desirable to re-examine the blood of birds. I accordingly worked on very large quantities of the blood of the turkey, goose, duck, and fowl. After the most careful examination I have never been able to detect any uric acid in the blood of these birds, and I therefore confirm Garrod's observations." "How is it possible (he asks) in all these cases that uric acid could be absent from the blood if the view be correct that it is formed in the system generally, and is conveyed in that fluid to the kidneys, which play, as it were, merely the part of a filter in the removal of uric acid from the blood. I consider that the evidence brought forward renders such a view impossible." He subsequently proceeds to formulate certain conclusions, those of importance from our present point of view being—

1. Uric acid is not normally present in the blood of man and other mammals, nor in the blood of birds.

2. Uric acid is normally produced only in the kidneys.

3. The presence of uric acid in the blood in gout is due to its deficient excretion by the kidneys, and to the subsequent absorption of the non-excreted portion into the blood from these organs.

These conclusions, in the main, agree with those formulated by Sir A. Garrod, and (if correct) they are of the highest importance in connection with the problems of gout. On theoretical grounds I felt unwilling to accept them, and also, especially in view of the fact that more than one continental authority had affirmed the contrary. I am also aware that modern physiological teaching does not include the formation of uric acid in the list of vital processes performed by the kidney; but in the absence of direct and positive proof of its production in other tissues, we want, if possible, conclusive evidence of its non-production in that organ. I accordingly made an extended series of observations on the blood of birds, a snake, and of various mammals. The fullest details as to the quantities of blood used, methods employed, etc., are elsewhere described,[1] and need not be detailed here. A few of the more important results may be given.

*Birds.*

| | | | Total Uric Acid. | Percentage. |
|---|---|---|---|---|
| Obs. 1 | Duck . | 1,750 c.cm. | 0.0352 gram. | or 0.0020 gram. |
| „ 2 | Goose . | 1,650 „ | 0.0428 „ | or 0.0025 „ |
| „ 3 | Turkey . | 1,500 „ | 0.0328 „ | or 0.0021 „ |

*Note.*—50 c.cm. of duck's blood were also examined and gave a positive result as regards the presence of uric acid. The sediment obtained was exceedingly scanty, but showed microscopically a few lozenge-shaped and rhombic crystals, which gave a distinct murexide reaction. No further examination was available.

*Snake.*

Blood and Heart.—On account of the very small quantity of blood present in the snake, it was considered advisable to remove the heart and large vessels with the contained blood and examine as a whole. A very fine deposit was obtained, which gave a distinct murexide reaction. The amount, calculated from the nitrogen obtained, was 0.0031 gram, including 0·6 mg. from the wash water used.

These results then, to my mind, conclusively indicate that the view advanced by Garrod and Luff as to the absence of uric acid from the blood of birds is an erroneous one, and the

[1] *Brit. Med. Journal*, Jan. 28, 1899, with Bibliog. references.

practical application of this statement to their views on the etiology of gout is at once apparent.

With the object, however, of testing the case further, I then made a series of investigations on the presence of uric acid in the livers, kidneys and spleens of various birds, on a snake and various mammals. The following conclusions were arrived at :—

1. Birds and snake.—The absolute amount in the liver was greater than that in the kidney.

2. Mammals.—(1) The absolute amount in the liver was greater than that in the other organs ; (2) The kidneys contained less than the spleen, both as regards absolute amount and percentage ; (3) The spleen seemed to contain a higher percentage than the other two organs.

These results then, to my mind, still further indicate that the views advanced by Garrod and Luff as to the kidney being the main seat of formation of uric acid are erroneous, and from that one readily deduces the fact that any conception of the etiology of gout based on this view is not to be entertained.

The next point appealing for investigation, was the examination of various pathological tissues and fluids. It is well known that a condition of uratæmia exists in certain diseases which have no ascertained relationship to gout, but it seemed advisable to make some detailed observations on the subject. The following are illustrative examples :—

| Disease. | Sex. | Age. | Fluid.[1] | Uric Acid. |
|---|---|---|---|---|
| Chronic Bright | F. | 60 | 10 oz. | ·0068 grm. |
| Malignant disease | M. | 55 | 4 ozs. Blood<br>26 „ Serum | ·0155 „ |
| Aneurism (aortic) | M. | 40 | 19 „ | ·087 „ |
| Ulcerative Endocarditis | M. | 30 | 26 „ | ·0075 „ |
| Lobar Pneumonia | M. | 22 | 10 „ | Qualitative reaction |
| Do. | M. | 40 | 16 „ | ·0075 grm. |
| | | | 17 „ | .0095 „ |

[1] This fluid was obtained from the pleural cavity after removal of the thoracic viscera and was usually diluted blood.

| | Liver.[1] | Spleen. | Kidney. | Lung affected. |
|---|---|---|---|---|
| Aneurism | ·1064 grm. | None | None | |
| Pneumonia (Croupous) | — | None | None | .0130 |
| Do. | ·1447 „ | None | None | |
| Granular Contracted Kidney | ·0386 „ | None | Qual. reaction | |
| Leucocythæmia[2] | (Weight of Spleen) $7\frac{1}{2}$ lbs. | ·0195 | | |

Can any deductions be drawn from these figures? Looking specially at the former set, those dealing with the blood, before formulating any conclusions, we must bear in mind at least two points of importance. In the first place, the amount of blood examined was only a part, and often a very small part of the total quantity, and further, we are dealing with the amount of uric acid present only at the particular time of examination. Keeping these points in view then, it seems clear that there is no absolutely close relationship between uratæmia *per se* and gout. In short the degree of causal relationship between them is, in general, over-estimated, or perhaps to put it more correctly, writers have failed to sufficiently observe the fact that in gout the error in proteid metabolism which induces uratæmia may also induce the presence in the blood of other bodies more or less toxic in character, which may be responsible for many of the symptoms of chronic or irregular gout. We have already seen one important point of positive evidence in support of this, viz., the condition of leucocythæmia, where there is a great excess of uric acid in the blood, without the development of uratosis or the accompaniment of the clinical manifestations of gout.

These points require a little further elaboration. If we wish to ascertain the extent to which the symptoms encountered in gout are due to the presence of retained uric acid we must pause

[1] The amount of Uric Acid found in these and other instances was very small, and is in marked contrast to the large quantities described by Haig as a result of his examination in similar cases.

[2] Numerous observers have affirmed the absence of uric acid from the Leucocythæmic spleen. Scherer is the only observer that has found it present. See *Brit. Med. Journal*, 28th Jan.

to think of a condition like leucocythæmia, where the blood is impregnated with it to an extent beyond that commonly seen in gout, and yet we neither see the clinical phenomena which are commonly supposed to be due to the uratæmia in gout, nor yet the uratosis which is the essential feature of the condition. Now, as uric acid is exactly the same compound under these and all other circumstances, it clearly follows that it is not the only important factor in gout. The nitrogen metabolism must be markedly different in the two conditions, and associated with that faulty nitrogen metabolism in the gouty individual which tends to the production of uric acid in excess, other bodies, about which we yet know little or nothing are probably produced, which may play a very important rôle in the production of the clinical manifestations of gout. Uric acid is only one of the direct products of proteid metabolism, and, while it may be a useful gauge of the extent of faulty metabolism, yet we are, I think, apt to err in looking so much to it alone. In leucocythæmia we have none of the uratosis which is an essential feature of gout. This deposition of uric acid in the tissues most depends on one of two things—(*a*) some alteration in the solvent power of the tissue fluids; (*b*) some alteration at the seat of deposition, or both of them in combination; neither of these conditions being present in leucocythæmia.

In our observations of the development of the minor and major clinical phenomena of gout we must study that development in its relationship to factors which influence these two factors. Specially is this so with DIET and DRUGS. These are questions into which I am not now entering, but a present reference to them is advisable. Much work is at present being done on uric acid metabolism and excretion after ingestion of certain articles of diet rich in "nucleins" and other substances which are supposed to be precursors of uric acid. Much more, however, requires to be done before we are in a position to speak on the subject except with the utmost caution. Of late there is a growing tendency to make much of the close relationship that exists between nucleins, uric acid, and leucocytosis. Thus we find Ewart, in his recent work on gout, saying: "An increased uric acid excretion goes hand in hand with an increase in the number of leucocytes, whether physiological, as in infancy or childhood and after ingestion of nucleins, or pathological as in leucocythæmia, pneumonia, etc." Such an opinion finds some

support doubtless from the combination of leucocytosis with increased uric acid excretion that occurs during digestion, but from many other standpoints lends itself to adverse criticism. I shall only refer to one of them, based on observations made by Milroy and Malcolm (*Journal of Physiology*). They, writing about the phosphorus and nitrogen excretion (the two known main products of nuclear disintegration) in a case of leucocythæmia, compared the results with a healthy man, and found—

| | White Corpuscles. | Phosphorus. | Uric Acid. |
|---|---|---|---|
| Normal . . | 8,000 | 4·058 grms. | 0·252 grms. |
| Leucocythæmia | 330,000 | 0·915 „ | 0·202 „ |

This then is conclusive evidence that there is no definite ratio between the number of leucocytes in the blood and the amount of uric acid in the urine. It also serves as corroborative evidence of the statement previously made, that the nitrogenous metabolism in the two conditions is essentially different. The secret of gout is the secret of nitrogenous metabolism. Until this latter is manifested, the former will remain unknown. Clinicians have opportunities in the careful observation of what seems to be noxious articles of food or drink or beneficial therapeutic means of studying metabolism in a manner more practical than scientific; but the results of their observations, when carefully made, are of quite equal importance with those made from the more purely physiological standpoint. The two methods of inquiry must go hand in hand. The subject is one of great complexity, and the difficulties that are in the way of our arrival at the real truth of it should be kept in mind. These are at least two in number. On the one hand we must realise that the problems raised by it invade the region of the unknown in physiology, on the other more practical and clinical side, it has to be borne in mind that a marked irregularity in the presentation of symptoms, alike as to their times of occurrence and degree of severity, forms a part of the natural history of gout. This latter point especially has to be borne in mind by the practitioner when he seeks to define the value of the dietetic alterations and therapeutic remedies that he adopts.

The results of these observations may be formulated as follows:—

1. From the comparative frequency of uratæmia, in the

absence of manifestations of gout, uric acid is not the all important factor that is commonly supposed.

2. The greater amount of uric acid in the liver than in the other two organs seems to indicate that the metabolic processes there are more conducive to uric acid formation, therefore the part played by it in the production of gout is a more important one. This is in accordance with clinical experience.

3. Any views as to uric acid excretion having a definite and constant relation to the number of white blood corpuscles in the blood, are not to be entertained.

4. In view of the conclusions arrived at by Moore as to the occurrence of uratosis, it would appear that that condition plays a more important part in our hospital cases than we are usually led to suppose.

In conclusion, I must express my indebtedness to the various hospital physicians who have kindly allowed me to utilise their material, and to Dr Milroy, Lecturer on Physiological Chemistry, Edinburgh University, for much valuable assistance throughout the course of the investigations.

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## Meeting VII.—May 3, 1899

SIR JOHN BATTY TUKE, *President*, *in the Chair*

### I. ELECTION OF MEMBERS

F. Reid Mackay, M.D., F.R.C.S.Ed., 5 Walker Street, Edinburgh, and Thomas Paul Gray, M.D., 6 St Catherine's Place, Edinburgh, were elected Ordinary Members of the Society.

### II. EXHIBITION OF SPECIMENS

*Dr R. A. Fleming* exhibited (*a*) a GUMMA OF THE BRAIN from a man aged forty who died under Dr James' care in the Royal Infirmary. The gumma was about the size of a walnut and was situated in the left Rolandic region near the point of division of the fissure of Sylvius. It had caused paresis of the right arm and leg during life.

(*b*) TYPHOID ULCERATION OF THE INTESTINE in a suckling aged ten months. Dr Fleming stated that no history to account for the condition could be obtained and death was due to peritonitis, resulting from operation for the relief of intestinal obstruction not associated with the typhoid condition.

(*c*) Two kidneys showing typical WAXY DEGENERATION.

## III. Original Communication

# AN OUTBREAK OF TYPHUS FEVER

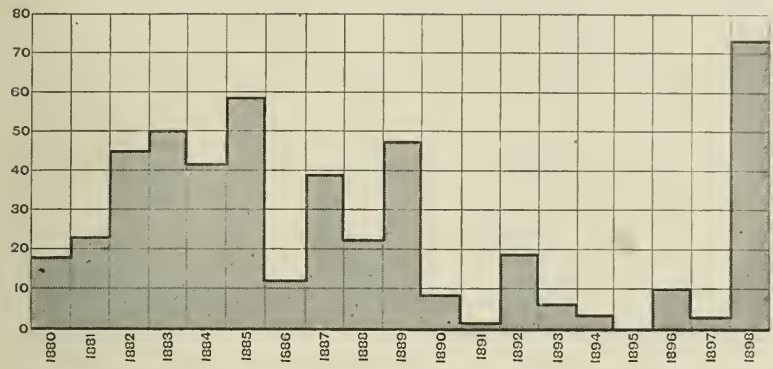
*History*—By Harvey Littlejohn, M.A., M.B., B.Sc., F.R.C.S.Ed.
*Clinical Features*—By Claude B. Ker, M.D., M.R.C.P.Ed., Superintendent, City Fever Hospital, Edinburgh.

History.—Murchison in his classical account of the continued fevers makes the remark that a complete history of typhus would be the history of Europe during the last three and a half centuries. With equal truth it may be said that no history of Edinburgh, of its development and of the conditions of life which existed up to a period within the memory of us all, could be written without frequent reference to this disease, which was at one time practically endemic in the Old Town; and annually desolated many homes, not only amongst the poor but also in the ranks of our own profession. Owing, however, to the general increase of trade and the improved social condition of the working classes, which higher and more regular wages produced, as well as to the vast progress which has been made within recent years in all that affects the health of individuals and of communities, epidemics of typhus fever have practically disappeared, and even an outbreak such as we propose to describe must be accounted an exceptional event in the sanitary history of the city.

Accurate information in regard to the prevalence of the disease, previous to the introduction of the Edinburgh Notification Act of 1879, is impossible to obtain, but the following table taken from Murchison gives the number of patients admitted into the Royal Infirmary during ten years, 1862-1871; and when we remember how few of the actual number of cases of the disease could have been treated in that institution, an idea is afforded of how widespread the infection was a quarter of a century ago.

| | | | |
|---|---|---|---|
| 1862 . . . | 14 | 1867 . . . | 303 |
| 1863 . . . | 74 | 1868 . . . | 280 |
| 1864 . . . | 212 | 1869 . . . | 259 |
| 1865 . . . | 447 | 1870 . . . | 287 |
| 1866 . . . | 847 | 1871 . . . | 101 |

Since 1880, however, we have accurate records, and in the table below are given the number of cases and the mortality




for each year, while the chart indicates even more clearly the history of the disease during the last nineteen years.

| Year. | Typhus. | | |
|---|---|---|---|
| | Cases. | Deaths. | Rate per cent. |
| 1880 . . . . | 18 | 7 | 39·0 |
| 1881 . . . . | 23 | 14 | 60·8 |
| 1882 . . . . | 45 | 10 | 22·2 |
| 1883 . . . . | 50 | 16 | 32·0 |
| 1884 . . . . | 42 | 16 | 38·0 |
| 1885 . . . . | 58 | 10 | 17·2 |
| 1886 . . . . | 12 | 4 | 33·3 |
| 1887 . . . . | 38 | 11 | 28·9 |
| 1888 . . . . | 23 | 5 | 21·7 |
| 1889 . . . . | 46 | 9 | 19·5 |
| 1890 . . . . | 7 | 1 | 14·3 |
| 1891 . . . . | 1 | ... | ... |
| 1892 . . . . | 18 | 3 | 16·6 |
| 1893 . . . . | 6 | 1 | 16·6 |
| 1894 . . . . | 3 | 1 | 33·3 |
| 1895 . . . . | ... | ... | ... |
| 1896 . . . . | 10 | 3 | 30·0 |
| 1897 . . . . | 3 | 1 | 33·3 |
| 1898 . . . . | 79 | 9 | 11·3 |
| Totals . . . | 482 | 121 | 25·1 |

During the ten years, 1880-1889, there were 355 cases notified, and in no year was there ever less than ten cases, the

average number being thirty-five. During the subsequent nine years, 127 cases have been notified, being an average of fourteen yearly; while, if we discard for the moment the seventy-nine cases which occurred last year, the average number of cases from 1890 to 1897 was only six, and only twice did the annual number of notifications reach double figures.

A still more remarkable contrast between the first period of ten years and the last of nine years is seen if we compare the death-rates of these two periods. From 1880 to 1889, 102 deaths occurred, equal to a death-rate of 28 per cent.; while in the subsequent nine years there were only nineteen deaths, giving a percentage of fourteen, or exactly half the death-rate for the ten years, 1880-1889. As a result of this gradual disappearance of typhus fever, the younger generation of medical men have little or no acquaintance with the characteristic features of the disease, and few of the students who have graduated in Edinburgh during the last twenty years can have had the opportunity of becoming practically conversant with its clinical aspects. That such want of knowledge may have disastrous results, is exemplified by the experience of last year's outbreak, and by many small outbreaks which have occurred recently in various parts of the country. For this reason, and also because the recent cases present some features of special interest, we venture to bring before you the following account.

In the first week of October four cases were sent into the City Hospital suffering from what was thought to be typhoid fever, although in one case the diagnosis was stated to be doubtful. All of the cases came from the neighbourhood of Simon Square, and resided within a hundred yards of each other. In the following week three cases were admitted to the Fever Hospital, two of them from public hospitals, and all resided in the same neighbourhood. One had been notified as a case of typhoid, another as scarlet fever, while in the third the suspicion of it being typhus was mentioned. Unfortunately, all of these patients had been ill for some time, and presented only fading rashes, but their whole appearance raised the suspicion that they were probably cases of typhus, and accordingly they were placed under observation in a separate ward. During the second week this suspicion became confirmed, owing partly to the localised distribution of the cases

and to the negative results of Widal's reaction. Much valuable time had been lost through the failure of the medical attendants to recognise the disease, and all of the first seven cases were only received into the Fever Hospital when the disease was well advanced, while three of them had been sent in the first instance as patients to other hospitals and kept there for some days.

As a result of this delay, a much wider dissemination of infection occurred than might otherwise have taken place, and the number of cases increased during the remainder of October and in November. During December they again decreased, the patients during this month coming for the most part from already infected houses, and by the middle of January the disease had completely disappeared.

The following table shows the number of cases which occurred during each week :—

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Week ending | | | October | 8 | . | . | 4 | cases. |
| „ | „ | „ | | 15 | . | . | 3 | „ |
| „ | „ | „ | | 22 | . | . | 4 | „ |
| „ | „ | „ | | 29 | . | . | 4 | „ |
| „ | „ | November | | 5 | . | . | 13 | „ |
| „ | „ | „ | | 12 | . | . | 6 | „ |
| „ | „ | „ | | 19 | . | . | 13 | „ |
| „ | „ | „ | | 26 | . | . | 12 | „ |
| „ | „ | December | | 3 | . | . | 5 | „ |
| „ | „ | „ | | 10 | . | . | 6 | „ |
| „ | „ | „ | | 17 | . | . | 4 | „ |
| „ | „ | „ | | 24 | . | . | 5 | „ |
| „ | „ | „ | | 31 | . | . | ... | „ |
| „ | „ | January | | 7 | . | . | 2 | „ |
| „ | „ | „ | | 14 | . | . | 1 | „ |
| | | | | | | | 82 | |

*Origin and distribution of the cases.*—A "de novo" origin of the disease may at the end of the nineteenth century be at once discarded, and if one fact was more clearly brought out by our investigation of the present cases than another, it was that practically the disease is only communicated by direct contagion, and that even persons who are residing in the same tenement with an infected family, and who are living under

conditions most favourable to receive infection, namely, dirt, squalor, and intemperance, do not become infected unless they have been in close contact with the patient.

It is true that we are unable to point to the first case of the disease in the City or explain how it was introduced, but this is not to be wondered at, when we consider the kind of life led by the lowest class of the population, their wandering habits and chance acquaintanceships. The time during which the disease must have existed before its true nature was recognised was also a serious obstacle to the discovery of its origin. The presence of the disease in many towns and districts of Scotland about this time is, we hold, sufficient ground for maintaining that the infection was almost certainly introduced from outside into the city, without being compelled to fall back upon a "de novo" theory.

The disease had probably existed for several weeks before it was recognised in October. The grounds for this presumption are the simultaneous occurrence of several cases at the beginning of October, and also the information which was elicited in our investigations, that one or two cases of severe "influenza," which confined the patients to bed and left them very weak, had occurred in the families which furnished the first true cases of typhus. It appeared also that a man residing in one of the worst parts of the infected area died at the commencement of September of "pneumonia," and that a wake which lasted for two days was held in connection with the body. The house where this occurred was small and of the most wretched description, and, according to the account of some of those present at the wake, the room was continually crowded. There is reason for suspecting that the man died of typhus fever, but whether this is so or not, it is a curious coincidence that at least three cases of so-called influenza occurred shortly afterwards amongst those who had been present at the wake, and later on three others were struck down with undoubted typhus.

As regards the *distribution* of the cases in the city, with a few exceptions they occurred within a radius of a quarter of a mile of Simon Square, and amongst families who were in constant intercommunication.

The cases which occurred outside of this area were for the most part clearly traced to have received the infection from

persons residing within it, but in one or two instances, notably a series of four cases in one family residing in Portobello, no direct communication could be discovered. The man was a hawker, and could give no very clear account of his movements or those of his family, so that the possibility of the disease being connected with the Edinburgh cases could not be excluded.

*Age incidence.*—The following table shows the ages of those attacked, and the remarkably large proportion of children affected must strike any one acquainted with previous statistics. Children under 10 years of age constitute over a quarter of the total cases, and are more numerous than the patients at the two age periods which usually supply the greatest number of attacks, namely, 15 to 20, and 20 to 25 :—

| | |
|---|---|
| Under 5 years . . . . . | 7 |
| 5 to 10 „ . . . . . | 15 |
| 10 to 15 „ . . . . . | 6 |
| 15 to 20 „ . . . . . | 8 |
| 20 to 25 „ . . . . . | 11 |
| 25 to 30 „ . . . . . | 8 |
| 30 to 40 „ . . . . . | 13 |
| 40 to 50 „ . . . . . | 10 |
| 50 and upwards . . . . . | 4 |
| Total . . . | 82 |

As regards *sex*, the cases were pretty equally divided, 38 males and 44 females being attacked.

*Mortality.*—Ten cases, or 12 per cent., ended fatally. This is a very low death-rate compared with the mortality given in connection with former epidemics, and I think it must be attributed, in part at any rate, to the fact that all the cases were treated in hospital, with the exception of two which were too ill to remove, and afterwards died. No death occurred under 15 years of age ; indeed, the predominating factor in regard to prognosis was not so much the age of the patient, as the existence of a previous life of intemperance.

*Infectiousness.*—Typhus is very infectious under certain favourable conditions, but its "striking" distance is very short. This point was repeatedly brought before us by the fact that a patient in a crowded tenement was not a source

of danger to other families, so long as members of these households did not actually enter the room where the patient was lying. In all the cases where the source of infection was traced, the patient was found to have been in actual contact with a previous case, while, without exception, families living in a common stair, in which there was an infected house, escaped the disease so long as no direct communication with the patient took place. Members of the patient's family might mix with other families in the stair or outside the house, apparently with impunity. This is an interesting fact, but it must not be used as an argument against the isolation of all cases of the disease in hospital, since in the class of the population amongst which typhus fever occurs, visits from relations and friends to the infected house are constantly taking place, and are difficult to prevent.

An illustration of this was afforded in a striking manner by what occurred to the following family :—

One of the first cases in October was a woman who lived along with her husband and family of six children in Paul Street. Little information could be obtained as to the existence of any friends or relations, who might have visited the infected house, and whom it was therefore important to keep under observation. The required information was, however, soon forthcoming by other means, as the following family tree indicates, and the dissemination of infection to which this patient gave rise was the means of discovering the intricacies of a family relationship, of which even the members themselves had lost count.

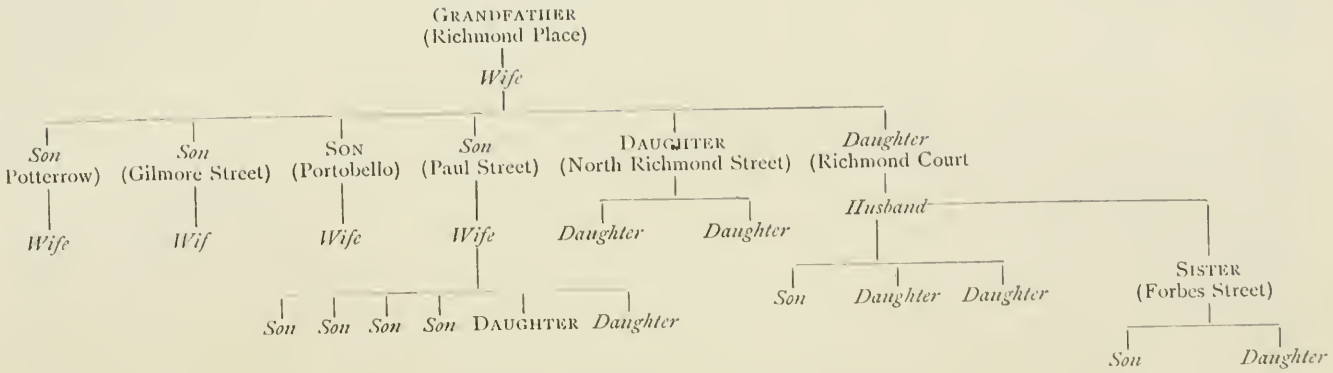
The wife infected her husband, and five of their children subsequently suffered from the disease ; the husband's mother, living in Richmond Place, the families of his three brothers and of two sisters, all residing in different streets—one of the former lived as far off as Portobello—were all infected, as well as the family of a sister-in-law.

In all, twenty-two members of the family suffered from typhus fever, and seven died. They resided in eight different tenement blocks, each crowded with inhabitants, and yet in one instance only did another family living in the same tenement become infected, and in this case it was fully proved that this was the only family in the tenement which had entered the infected house, and was on friendly terms with the inmates.

Dirty houses, deficient cubic space, and want of ventilation greatly increase the infectiousness of typhus; while, under the opposite conditions, the risk of anyone taking the disease from a patient is much reduced. In support of this proposition, it was observed that the great majority of the cases occurred amongst people who were living under the most insanitary conditions of overcrowding and uncleanliness, and that the fever was much more frequently spread to others from these cases than it was from the cases which occurred in clean and well-ventilated houses. The experience of the fever hospital also bears this out. Notwithstanding the fact that seventy-eight cases were treated there, not one of the staff, who came into contact with the cases, caught the disease, a result which is at variance with the usual experience of hospitals where typhus fever is treated, and which must, we think, be ascribed to the great care bestowed upon the personal cleanliness of the patients, and to the free ventilation maintained in the wards.

That "fomites" may convey the infection must be admitted, although they are not nearly so active in this direction as is commonly supposed. This was shown by the apparent absence of danger to those who mixed with inmates of infected houses in tenement buildings, and the non-conveyance of infection by persons residing in infected houses, to those with whom they came in contact at their work and elsewhere. That fomites, however, may convey infection, was proved by the case of one of the inspectors of the health department, who contracted the disease from a mattress which he cut up, preparatory to burning it, and which had been taken from a very dirty house, from which a patient had been removed. This inspector had not been exposed to infection previously, and he sickened with the fever thirteen days afterwards.

*Preventive measures.*—The means which ought to be adopted to prevent the dissemination of the disease are—(1) Isolation of the patient in hospital.—This is essential under all circumstances. (2) All persons living in the same house, and all friends or neighbours who have been in close communication with the patient, should be removed into quarantine for fifteen days.—This is the best means of stamping out the disease quickly, and as a rule, amongst the class of people with which one has to deal, little objection is offered to this procedure, provided some small compensation is given to cover the house rent, and an arrangement is




Italics denote cases of typhus fever.

effected whereby the men do not lose their employment during their detention. If removal to quarantine is not carried out, then all persons who have been exposed to infection should be frequently visited at their homes, so as to discover as soon as possible any symptoms of illness appearing amongst them. In many instances where the people could not be taken into quarantine they were removed for a few hours to hospital, while their houses were disinfected, and they themselves received a bath, and had their clothes passed through the steam disinfector. (3) Disinfection.—This should be effected by removing all textile articles from the house and disinfecting them by steam—or, in the case of old mattresses, burning them—thoroughly fumigating the rooms with sulphur, or, better still, spraying them with formalin or perchloride of mercury solution; and, lastly, by the plentiful use of soap and water, fresh air, and the whitewashing of the ceilings, stairs, etc.

The infective agent of typhus does not appear to be difficult to destroy, and even free exposure to fresh air alone seems to have a great effect in inhibiting the spread of infection. In none of the houses which were disinfected, according to the above method, was there a recurrence of the disease.

The Clinical Features.—The whole subject of typhus has been so completely and admirably treated in the great work of Murchison, and has been so thoroughly summarised and brought up to date in Moore's book on the infectious fevers, that I feel it is necessary to apologise before entering on this paper. The only excuse that I can offer is, that it is so long since we have had so many cases together in Edinburgh, that it may be of some interest to describe the fever as we saw it in the wards of the City Hospital, and see if there has been any change in its characteristics since the time when it was endemic in the Old Town.

It is not in my province to speak of the predisposing causes of typhus; and of the exciting cause, almost certainly a micro-organism, we as yet know absolutely nothing. Various germs have been isolated by different observers without any absolute proof that they are the cause of the disease. During the recent outbreak, two former assistant medical officers of the hospital were good enough to undertake a bacteriological

research ; but though they have had some very interesting results, it is too early to say if they are to be fortunate enough to clear up the mystery which at present enwraps the origin of the fever.

*Incubation period.*—It is generally admitted that the period of incubation in typhus may vary considerably in length, being in some instances only a few days, whereas in others it may be prolonged for nearly three weeks. During the recent outbreak the patients have for the most part lived in such intimate relations with each other, and have been exposed on so many occasions to the contagion, that it is absolutely impossible in the vast majority of the cases to define the length of the period. In one instance, however, the patient had only one chance of exposure, and his first symptoms occurred thirteen days afterwards. One of our ambulance attendants fell ill twelve days after destroying infected fomites. Another case took over seventeen days to develop his fever. During this time he was kept in quarantine, and certainly was not exposed to infection. On the other hand, though the statutory period of quarantine adopted was only fourteen days, and many persons were discharged after that interval, none of them returned to us with the disease.

*Invasion period.*—The onset of typhus is very sudden, and

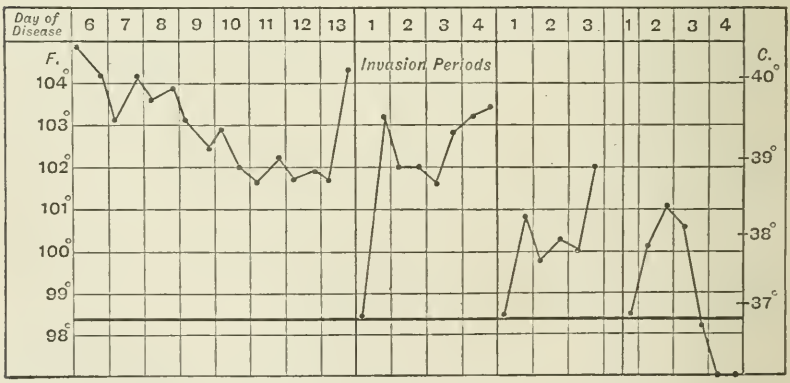



FIG. 1.—A, a fatal case ; B and C, showing different forms of invasion (illustrated also by Figs. 2 A, and 4) ; D, chart of an infant exposed to the infection.

the patient can as a rule tell not only the day but even the actual hour of his attack. The first symptoms to be complained of are usually severe headache, and a feeling of chill

often accompanied by actual shivering. Pains in the limbs and back are also frequently felt, and at least two of our cases were from this cause diagnosed as rheumatic fever. It is usually stated (Murchison and Moore) that, while nausea is frequent, vomiting is rare. It is therefore of some interest to note that in my cases vomiting was a quite common symptom, occurring in three-fourths of the children and in nearly half the adults. Rigors were not very common. As regards the relative frequency of the other symptoms, it may be said that headache was practically invariable; that a feeling of chilliness was noted in 80 per cent. of the cases; and, lastly, that the three symptoms —headache, chilliness, and vomiting—were combined in 37 per cent. of the admissions.

The temperature commences to rise at once, and in most cases reaches the acme in three days. Occasionally there is a sudden rise to a high level on the first evening, but this only occurred in two out of eleven cases watched from the inception of the disease, all the others mounting comparatively slowly. In the case of two children and of one adult there was actually a fall to normal on the second day.

Even at this early stage the patient often may be diagnosed by his appearance. The face is usually conjested and stupid, while the eyes are pink and watery. Great depression is always felt, and it is quite exceptional for a typhus patient to keep on his feet after the third day.

*Period of advance and eruption.*—This period may be said to start on the fourth day. The patient has taken to his bed, and suffers chiefly from headache, thirst, and insomnia. The temperature in an average case is 103° or 104°, and the pulse is accelerated in proportion to this elevation. The tongue, which during the period of invasion has been covered with a close, white fur, now becomes dry and yellow, and sordes appear on the lips and teeth. There is always great confusion of ideas, and the patient suffers from deafness. There is often slight delirium at night. As a rule, the bowels are constipated.

The rash in my cases was usually well out by the fourth day. It may be said to consist of three main elements. First, spots appear on the surface of the skin. These spots are at first very like those of enteric fever. They are rose-pink in colour, and fade easily on pressure, and for the first few hours

of their existence are distinctly raised above the skin. After they have been out for a couple of days, they become a dirty brown in colour, and do not disappear on pressure. In distribution this rash is present all over the body except the face. The spots are somewhat irregular in outline, and give a measly appearance to the patient. The second element of the typhus eruption consists of similar spots lying, not on the skin, but faintly seen as it were beneath it, and causing what is known as "subcuticular mottling." This is best seen on the trunk, and perhaps best of all in the axillæ. Lastly, true hæmorrhage may occur into either the cuticular or subcuticular spots, giving rise to dark purple petechiæ, exceedingly like flea-bites, but lacking the central point.

While it is usually stated that the rash is not visible before the fourth day, I noted that the subcuticular element was as a rule present in my cases on the third, and in one or two instances quite recognisable, though very faint, on the second. It was present more or less distinctly in all my adult cases, and was observed in all the children except two. Unless the petechial element is very marked, the rash begins to fade in the second week of the fever, though staining occasionally persists for some days after the crisis.

At the end of the first week the patient is apt to suffer from restless delirium. He appears very drunk, and probably tries to get out of bed. In severe cases, especially if they have been alcoholic, the excitement may be intense, the so-called delirium ferox setting in. It is this wild delirium which occasionally causes patients to throw themselves from windows, and when it has developed it is safer to use straps or some other method of restraint.

By the time the patient enters the second week of his fever, he is usually too exhausted to continue wildly delirious. He is now more likely to be lying helpless on his back and muttering only. If at all severe, he has subsultus tendinum, and picks at the bed-clothes. In this helpless state he drifts through the remainder of his fever, passing his evacuations under him, and often taking his nourishment only with difficulty. The face is now more drunken and bloated than ever, the eyes are blood-shot, and the pupils are contracted. The tongue, if not properly attended to, becomes crusted and black, and may be shrivelled and pointed. The pulse loses its tone, and is apt

to run. There is nearly always some hypostatic congestion of the lungs.

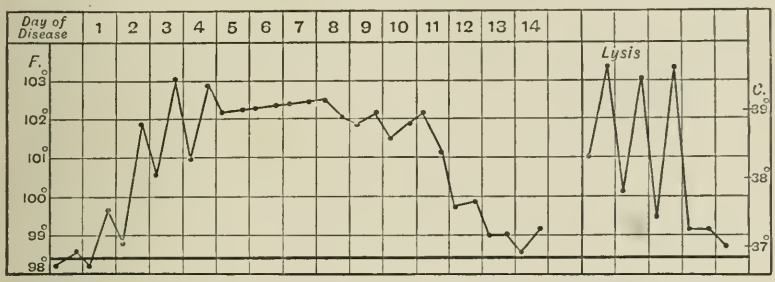



FIG. 2.—A, Complete course of fever in woman æt. 67; B, example of termination by lysis in a child.

*Stage of defervescence.*—Towards the middle or end of the second week there is often a considerable improvement. Cases which are going to recover seldom reach the fourteenth day without some amelioration. On the other hand, fatal cases get progressively worse during the second week, though most of my own cases did not die before the fourteenth day.

The termination of the fever may be expected about the thirteenth, fourteenth, or fifteenth days. The crisis is in some cases exceedingly well marked, but it is not as a rule very rapid. In this group of cases it never took less than two days to bring the temperature to normal, and in the majority took three. The improvement of the patients with the fall of temperature was very striking, and, as a rule, there was not much sweating during the process, nor was there much tendency to collapse. After the temperature fell it usually remained subnormal for nearly a week, and during that time the pulse remained slow and shabby, with a tendency to intermit. As soon as the fever was over, the patients took their food ravenously, and put on weight rapidly.

If, on the other hand, the termination is going to be a fatal one, the temperature instead of falling gently from the middle of the second week, remains level, or may show a tendency to rise. Six of the fatal cases died just at the fourteenth day, and with rising temperatures. The remaining three survived their crisis to die of exhaustion in a few days later.

While the above may be taken to be a rough sketch of the average course of the fever, it may be of some interest to give

a more detailed analysis of the cases of this outbreak. First, as regards the duration of the disease, the evening temperature

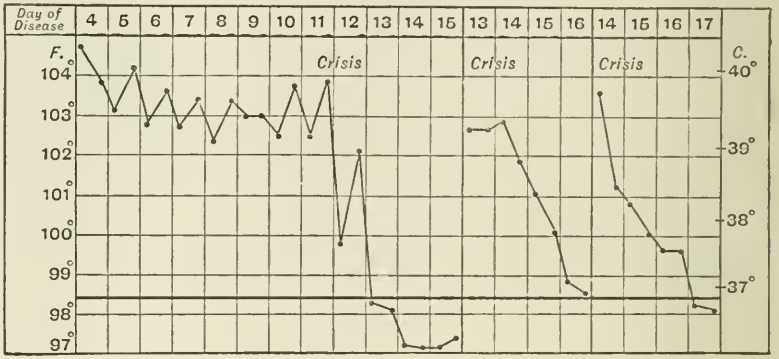



FIG. 3.—A, Chart of a girl of 14, with termination by crisis; B and C, examples of termination by crisis in adults.

of my adult patients became on an average steadily normal on the fifteenth day. The children ran a shorter course, attaining the normal on an average in eleven days and a half.

The nervous system is always severely affected in typhus, and my cases showed no exception to this rule. Delirium occurred in forty out of fifty-one adult cases. It was chiefly of the low, muttering type; but three cases, all males, and all of which ended fatally, had true delirium ferox about the end of their first week. Persistent insomnia was another very common symptom, complicating nearly half the adult cases. Subsultus tendinum was noted in over a third of the adults. Twitching of the muscles of the face occurred in six cases only, and in two of these there were actual convulsions of a choreic character, starting in the face and spreading to the upper extremities. Incontinence of urine was also frequently present, nineteen of the fifty-one adult patients suffering from this condition. Twelve adults showed retention of urine, and nearly all these required the use of the catheter for several days.

The urine in nearly all the adult cases contained albumin, thirty-eight out of the fifty-one having this complication as long as the temperature remained up. In only a few of these, however, was it present in any considerable quantity. Fifteen cases were examined for Ehrlich's diazo reaction, and every one of them gave it well, showing conclusively what little

reliance can be placed on that test as a differential diagnosis between enteric and typhus.

The bowels in all my cases were very constipated for the first week. About the tenth day, however, there is a slight tendency to looseness. This is a point of some practical importance, as one is very tempted to use purgatives, and one given at this juncture is very apt to set up an intractable diarrhœa. In fact, I am far more careful about the administration of aperients in typhus than even in enteric.

I was fortunate enough to have one case which illustrated the fact that it is possible to suffer from this fever twice. A woman, who was treated in the hospital six years ago for typhus, and who at that time undoubtedly had a severe attack of the fever, contracted it a second time, and walked into the hospital saying that she had it. We were a little incredulous, but the result proved she was right.

*Complications.*—Except for the very frequent occurrence of hypostatic pneumonia, the cases suffered from very few complications. Two women were admitted pregnant, at the eighth and fifth months respectively, and both recovered without abortion. Two women had true lobar pneumonia, and one of the fatal cases was complicated with a severe bronchitis. Three cases, all women, had otorrhœa, and this in one instance was followed by Bell's paralysis. In seven cases diarrhœa was serious enough to assume the importance of a complication. As regards sequelæ, one man had parotitis, another a sharp attack of nephritis, while boils and small abscesses of the nature of those which occasionally retard convalescence of enteric fever gave considerable trouble to three patients.

*Mortality and prognosis.*—Nine of my cases proved fatal out of a total of seventy-nine. This gives a mortality of nearly 11½ per cent., the exact figure being 11·39. Age, as all observers have agreed, seems to have a great influence on the death-rate. Thus of twenty-four individuals below 12 years of age none died, and of thirty-five under 20 years only one, giving a rate of 2·8 per cent. Of thirty patients from 20 to 40 years of age, four died, a rate of 13·3 per cent.; and the increase in the percentage of deaths is still more marked in the fourteen remaining cases of more than 40 years of age, of whom no less than four succumbed, the death-rate rising to 28·5 per cent., or more than double that of the preceding group.

As regards sex, females are admitted by most authorities to have a lower death-rate than males. Of the nine fatal cases which occurred in the City Hospital, seven were men. Murchison has suggested that the greater muscular development of men offered, as it were, more material for the disintegration caused by the fever process, or, as we might say nowadays, more pabulum for the unknown germ that causes the disease. He also pointed out that male patients were more likely to be alcoholic. I think my own few cases bear out this theory very well, as all the men who died were, without exception, large, heavy, and very muscular men, and five of them were distinctly alcoholic cases. The adult males that survived were, it is true, in many instances alcoholic, but only two or three of them were physically the equals of the seven who died. It would almost seem that a fine physique is a distinct disadvantage to a man starting an attack of typhus. Mere body weight appears to have something to do with it, as the most severe cases among the women were those who were fattest and heaviest. The figures relating to sex death-rates are as follows: Of thirty-eight males of all ages, seven, or 18·4 per cent., died; of forty-one females of all ages, two, or 4·8 per cent., died; whereas, if we take merely the adult cases, the percentage death-rate of males is 26 per cent., while that of females is only 7·14.

The prognosis of the fever, then, must be largely influenced by questions of sex and age. After this, probably the most important aid to prognosis is the condition of the nervous system. A patient who sleeps well, who has no subsultus tendinum, and who is able to be roused sufficiently from his lethargic condition to put out his tongue when asked, will be likely to recover. On the other hand, severe nervous symptoms, especially either wild delirium or coma, are of evil omen. The rash also gives a direct indication as to the severity of the case; profuse rashes, with many petechiæ, showing that the case is likely to be a serious one. Without being a very ardent believer in the old views about critical days, I am inclined to think that there is something to be gained in the way of prognosis by a careful study of the temperature about the seventh day. A very large number of my charts show a slight fall of temperature at that period, and some cases seem to start, as it were, a very slow lysis from that time to the thirteenth day, when the fall

becomes more abrupt. Such a gentle decline of the fever is always a good sign.

*Diagnosis.*—A history of exposure to the disease, a sudden onset and early prostration, accompanied by mental confusion, should, if there is an epidemic, suggest the fever. The congested face and red eyes are also very suggestive. The presence of the rash is of course final, but it may be exceedingly difficult to see in the dark rooms usually the home of typhus patients; and, again, a good wash is often required before it becomes visible. Many of our cases were sent in as enteric fever, and when the rash is badly developed that is the most natural mistake. To distinguish the two fevers, as I have mentioned above, there is no assistance to be got from Ehrlich's reaction. Widal's reaction is the best method to use if in doubt. I am quite satisfied that it is to be relied on. Previous to this epidemic I found it present in two cases of typhus out of ten examined. I believe that this apparent inconsistency is due to the cases in question having suffered from typhoid at some previous date. During the recent outbreak I used the reaction in all cases where a differential diagnosis was required. In all the ten cases of undoubted typhus the reaction was not present. There should be little difficulty in distinguishing the disease from measles, the history of or the presence of a rash on the face pointing to the latter. As regards acute lobar pneumonia, the differential diagnosis may be exceedingly difficult. In uncomplicated typhus there is often dulness at the bases of the lungs, but this is not restricted to the limits of the lobe, and tubular breathing is not present. The respirations, moreover, though accelerated, are usually only slightly more rapid than the temperature would account for. Perhaps the most difficult cases of all to differentiate are bad influenzas, and, as I have said above, acute rheumatism may have to be remembered.

No remarks about the diagnosis of typhus would be complete without some allusion to the characteristic odour of the fever. This odour has been compared to that of mice and also to that of rotten straw. I think personally that it is peculiar and unpleasant enough to be acknowledged as an odour *sui generis*. It appears to be given off from the skin of nearly all typhus patients at some period or other of the disease. Its development is, I am certain, entirely independent of the cleanliness of the patients, as I found it unmistakably present in

patients who had been three weeks and more in hospital at a time when they were well advanced in convalescence. It is of undoubted value in diagnosis, as, if it is noticed at all, it cannot possibly be mistaken for anything else. It is usually more appreciated by nurses than by doctors, probably because their sense of smell is not impaired by smoking. In two instances I was able to pick cases out of my enteric wards, which, having mild symptoms and very badly developed rashes, would have been missed altogether, had it not been that their very suspicious smell attracted my attention.

*Treatment.*—In an acute fever that runs a definite course there is seldom much to be done in the way of active treatment, and typhus is no exception to this rule. In the first place, care was taken to keep up the patient's strength. The liberal use of strong beef-teas and various meat-juices saved the hospital a considerable outlay on stimulants. The staple diet was milk, and this in some cases was supplemented by Benger's food. The milk was invariably given in measured quantities in the form of small meals every two hours, and cold water was given to, and indeed forced on, the patients between times. As soon as the patient had a normal temperature, fish, bread and milk, and eggs were allowed.

The question of stimulating in fevers is always interesting. Typhus is one of those that seems in most cases to require stimulants. While in every case meat-juices were first given a trial, nearly three-quarters of the adult cases and nearly a third of the children required alcohol at some period or other of their course. Whisky was used in each instance except in one case, where stimulants of all kinds were badly borne, and brandy and champagne were used without effect.

As to cardiac tonics other than alcohol, I cannot say that we found them of much service. Strophanthus in two or three cases seemed to do good, but was exceedingly disappointing in others. Strychnine, perhaps, had the best effect on the pulse, but increased the nervous excitement in several cases so markedly that it had to be stopped.

Nearly half the adult patients required one or more doses of a hypnotic. I found I got the best results by giving hypnotics early and freely. Waiting too long meant frequently failure of even a very heavy dose to act. Both sulphonal and paraldehyde were found useful, the former causing a much more

prolonged sleep, but often seeming to increase rather than diminish the ataxic symptoms of the patient. In the cases where there was much facial twitching I used chloral cautiously, and in one or two instances undoubtedly to good effect.

Great attention must always be paid to the condition of the bladder, especially in the case of delirious patients. A routine percussion of the organ twice daily will save a great deal of subsequent trouble with the catheter. When the use of an instrument becomes imperative, it is advisable to draw off the urine at least every twelve, and preferably every eight hours.

The temperature was in no case interfered with by drugs. Even in those cases which ran continuously at a level of 105°, the most that was done was the frequent use of tepid sponges. No case suffered from hyperpyrexia. This may have been possibly due to the fact that the wards were kept at a very low temperature, and the patients allowed only one sheet and one blanket as long as their temperature remained up. In addition to this, many of the sharper cases, if they were passing their evacuations under them, were left naked, in order to save them the exhaustion undoubtedly caused by the frequent changing of soiled night shirts. It is almost needless to say that there was no complaint of cold from patients suffering from high temperatures, and I am inclined to believe that this method of surface cooling has to a certain extent the effect that would be obtained by cold baths, while it is much easier to carry out. It is in fact the treatment by "the ambient air," recommended by de Souza as a substitute for baths in enteric fever. The ward temperature never exceeded 60° F., and was much more often 55° F. in the daytime, while at night it was frequently below 50° F. As the outbreak occurred in the winter, there was no difficulty at all in maintaining this low level, by keeping open all the doors and windows at all times. When the crisis had occurred, extra blankets were at once added to the bed, and it was usually found useful to wrap another blanket round the head and shoulders. As soon as possible, also, the patient was removed to a convalescent ward, kept at an ordinary temperature. I may add that there was no instance of a patient catching cold even under these somewhat severe conditions.

There were in all sixteen nurses exposed to the fever and four doctors. It is a great contrast from the old days of typhus epidemics in Edinburgh, to be able to report that none of these

took the disease. It is true that one of our nurses died of the fever, but, however she may have contracted it, she was never exposed to it in the wards. I attribute the immunity of the others to the very free ventilation I insisted on maintaining in the wards, and to the great care exercised in the supervision of their general health. They were advised to take particular care of their bowels, to take frequent baths, to secure a healthy action of their skin, and to get regular and frequent exercise in the open air. Any nurse who appeared to be losing her appetite was at once removed from the wards, but I am bound to say that, with the exception of the so-called typhus headaches, from which all of us who came in contact with the fever often suffered, the health of the staff remained very good. I may say here that colds, in spite of the chilly and draughty condition of the wards, were unknown, and that the nurses in no way seemed to suffer from the undoubted discomforts of the temperature to which I felt bound to expose them.

Another precaution, that of frequently sponging the patients with an antiseptic solution, may also have contributed to the safety of the nurses. Every case in the wards, whether acute or convalescent, was sponged twice daily with Jeyes' fluid diluted with water. This not only kept down the odour of the disease so well that it was impossible to appreciate it on entering the ward, but was probably also of some real value as a prophylactic. The toilet of the patients' mouths was also carefully attended to, swabs of Listerine and of an ointment of boracic acid and menthol being used every few hours.

In the wards set apart for quarantine I found it convenient to keep the patients in bed. This may seem at first sight somewhat of a hardship, but curiously enough I had not a single complaint, and therefore am justified in supposing that it did not fall very heavily on the persons secluded. The advantage of such a system depends on the certainty which it gives that any person who develops the fever has not been in actual contact with anyone else in the ward, the chances of the disease being transmitted from bed to bed during the few hours that elapse before the patient's removal being too remote to be seriously considered. The temperature of all persons under observation was taken regularly night and morning, and also at the first complaint of headache or malaise.

*The fever in children.*—An account of the outbreak would not be complete without some allusion to the course of this

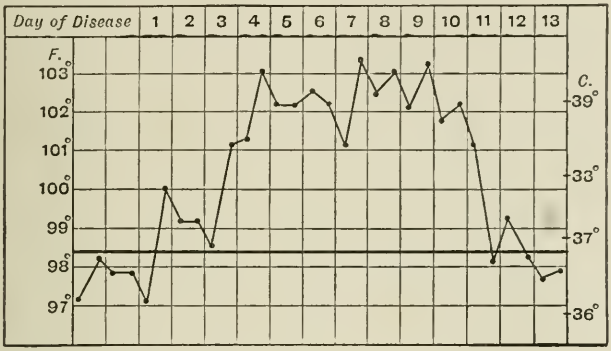



FIG. 4.—Chart of a child æt. 5, showing insidious invasion and short duration.

fever in children, especially as the number of children affected has been so high. It has been noted above that the mortality of the children treated was nil. And it may be said that the incidence of all the severer symptoms of the disease was much less than in adults. The temperature, though in some cases fairly severe, was in most subacute. The actual duration of the fever was less, several of the cases terminating by crisis about the seventh day. Others, though having good rashes and well-marked symptoms, terminated by a regular lysis, lasting in some instances five or six days. Not one had albuminuria, only two had subsultus, and not more than seven had delirium.

While I have only included in the above notes cases that were undoubted typhus, I had occasion to move two or three cases from the quarantine wards suffering from indefinite temperatures, which lasted only two or three days. There were practically no other symptoms, but I incline, rightly or wrongly, to the belief that the patients, all very young children, were in reality affected with an abortive form of the fever. Unfortunately, it is impossible to prove this theory. The fact that they did not contract the disease when transferred to the typhus wards goes for nothing, as, if they escaped it under the bad sanitary conditions of their own homes, it is highly improbable that they should catch it in our well-aired wards.

Before concluding this paper, I would like to acknowledge

valuable assistance received from Dr Littlejohn in the diagnosis of doubtful cases, and from Dr Wyllie, the consulting physician to the hospital, in the treatment and general management of the patients.

In conclusion, I think that the heavy death-rate among the adult male patients, the heavy incidence of the nervous phenomena, and the severity of the rashes, prove that this outbreak was at least of average severity, and that there has been no change of type since the days when the fever used to infest this city. This view was also taken by senior members of the profession who saw these cases, and who remembered the epidemics of old days. Under the circumstances, then, I think we have to congratulate ourselves, both on the rapidity with which it was stamped out by the public health authorities, and on the fact that none of the persons attending on it lost their lives.

## Meeting VIII.—May 17, 1899

SIR JOHN BATTY TUKE, *President, in the Chair*

The President, before commencing the business of the meeting, made suitable reference to the death of Dr John Moir, who had been a Member of the Society for the long period of sixty-three years.

### I. EXHIBITION OF PATIENTS

1. *Mr Shaw M'Laren* exhibited—

(*a*) A boy, aged 7, on whom he had, three weeks before, operated for CONGENITAL TORTICOLLIS. He mentioned two points of interest: (1) The operation, instead of the subcutaneous tenotomy, was the now more usual "open" myotomy, and the result was perfect without the use of apparatus except adhesive plaster and bandaging; (2) the case showed well the asymmetry sometimes seen, the distance from eye commissure to lip-commissure being about a quarter of an inch less than on the affected side.

(*b*) A lad on whom he had operated about eight months before for RUPTURED BLADDER, caused by a cart falling on him. The rupture was extraperitoneal, being near the urethra. The diagnosis was made from swelling above the pubes, pain, and

the passage of small quantities of blood and urine by the urethra and by the catheter; the injection test failed. Instead of attempting to stitch the wound, he made a counter-opening in the perineum and drained below and above the pubes. The convalescence was protracted, but the parts were now normal again.

(*c*) A man, aged 70, from whom he had removed a PAPILLOMA (NON-MALIGNANT) OF THE BLADDER. The only symptom was the occurrence, once, of a severe hæmaturia, which soon passed away. The cystoscope made the diagnosis of tumour easy. The bladder, at the operation, was distended with air from a cycle-pump instead of fluid. The incision was the vertical one, which gave ample room, though the position of the tumour was a difficult one.

(*d*) A patient with untreated POTT'S FRACTURE with marked displacement of the external malleolus backwards, and dislocation backwards of the foot, most of the astragalus lying well behind the tibia. Two skiagraphs were shown.

2. *Mr F. M. Caird* exhibited two patients who had been presented to the Society on a previous occasion.[1]

(*a*) A man, aged 68, who had suffered from CARCINOMA OF THE PYLORUS for which pylorectomy after the method of Kocher had been performed three years ago. He has since enjoyed perfect health.

(*b*) A man, aged 54, who three years ago was supposed to suffer from a similar affection, but in whose case the size of the growth and the complication of the neighbouring lymphatic glands precluded excision. Accordingly, gastro-enterostomy was performed. At the operation there was no doubt felt as to the malignant nature of the tumour. The patient rapidly gained in health and weight thereafter, and about eighteen months after the first operation he returned because of a ventral hernia which had recently developed at the site of the incision. To remedy this condition the abdomen was once more opened, when not a trace of the original growth or glandular infection could be discerned. The pylorus lay in the midst of soft pliant adhesions. The original condition was therefore presumably of the nature of chronic inflammation. His present state is excellent in every respect.

[1] See *Trans. Medico-Chir. Soc.*, Edin., Vol. XVI., p. 2 (*b*) and (*a*).

3. *Dr J. O. Affleck* shewed three cases of DIABETES—

(*a*) A boy, aged 14, with DIABETES MELLITUS in a very acute form. The history of the case was such as to indicate that the disease had existed for only about eight weeks, and the symptoms were advancing rapidly. The patient presented the facies of diabetes in a very typical manner. The urine varied from 150 to 250 oz. per diem, and the quantity of sugar kept pretty constantly at about 34 grains per ounce. Treatment had made no impression on the progress of the case.

(*b*) A man, aged 42, with DIABETES MELLITUS which had lasted for at least two years, but was now advancing rapidly. The amount of urine and the quantity of sugar were almost the same as in No. 1 ; but the case was complicated with optic atrophy and with well-marked peripheral neuritis.

(*c*) A boy, aged 16, with DIABETES INSIPIDUS which had lasted for about four years. The daily amount of urine was from 400 to 600 ounces, and its specific gravity was scarcely 1001. There was excessive thirst. The patient had a very diabetic appearance, but he had not emaciated much and was now rather increasing in weight. All the usual remedies had been tried without effect.

4. *Dr C. W. MacGillivray* exhibited two cases of PERFORATING GASTRIC ULCER successfully operated on :—

The two cases of perforating gastric ulcer which he had thought worthy of recording are specially interesting as proving that even in the most unfavourable conditions, prompt and thorough measures can lead to results more favourable than could possibly have been expected. The first was that of a housemaid, æt. 23, who was admitted to his wards on the evening of January 5th, 1899. Her history was as follows. For some time previously she had suffered from anæmia, with occasional gastric symptoms of indigestion and pain after food, but never severe enough to prevent her from doing her ordinary work. On the morning of January 4th she had considerable pain, but was able to continue work and take her food as usual. On the morning of the 5th the pain returned, and there was some sickness, in which was a trace of blood. She, however, went about as usual, ate a hearty dinner of broth, beef and potatoes, and took her tea at 4 P.M. She then dressed and went out, but while walking about 8 P.M. was

suddenly seized with intense abdominal pain and faintness, necessitating her being sent home in a cab. She was at once put to bed; hot fomentations were applied, and a doctor was sent for. He at once recognised the gravity of the condition and had her sent up to the Infirmary, where she arrived about 11.30. When he (Dr MacGillivray) saw her at midnight she was lying on her back in bed, somewhat pale, but wonderfully little collapsed; her pulse was 98 and her temperature 99·4; her legs were not drawn up. She complained of abdominal pain, especially on movement and on deep pressure over the epigastric region. The abdomen was moderately distended and rigid, the respiration thoracic. On percussion the note was tympanitic, there being no liver dulness. A perforated gastric ulcer was diagnosed, and, chloroform having been administered, an incision was made from the ensiform cartilage to the umbilicus. Gas and a thin flaky purulent fluid at once escaped from the peritoneal cavity. The stomach was brought up to this, and was found to be congested and covered with easily detachable flakes of lymph. Near the cardiac end of the anterior surface close to the lesser curvature there was a thickened and indurated patch, in the centre of which was an irregular perforation with sharp edges large enough to admit the tip of the little finger. An elliptical portion was excised including the perforation, and the edges were brought together with a continuous silk suture passed through all the coats of the stomach. Superficially to this, a series of interrupted fine silk Lembert sutures were inserted. The whole abdominal cavity was filled with an odourless, purulent fluid like thin peasoup, Douglas' pouch containing more than a pint. A counter opening was made above the symphysis pubis, and the cavity was thoroughly washed out with hot saline solution. The incision was then closed in successive layers, and glass drainage tubes inserted both supra-pubically and up to the seat of perforation. On removal to bed she was suffering from very little collapse. She had a good night, and next morning was comfortable and happy, there being about an ounce of purulent discharge on the dressings. Nutrient enemata were administered every four hours; the bowels moved, and flatus was passed. The second day milk was given by the mouth, and thereafter the course of the case was uneventful, except for the fact that the

upper wound opened slightly at the lower end, and for about a week most fœtid pus was discharged mixed with fatty shreds. She never had, however, any pain, fever, or discomfort, and left for the Convalescent Hospital, perfectly healed, rosy, and with a good appetite, at the end of February.

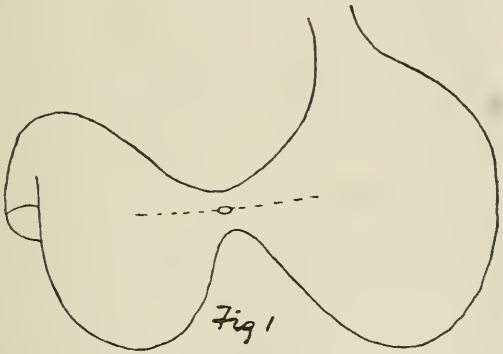
The special points of interest in the case are (1) that at so short an interval, at most five hours after the apparent time of perforation, the abdomen should have been filled with a purulent fluid; no doubt, however, leaking must have commenced on the morning of the previous day; and (2) that such marked conditions should have been present in the peritoneal cavity with such slight objective and subjective symptoms.

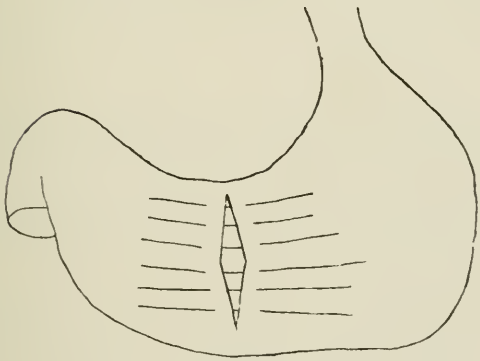
The second case was equally interesting.

A woman, æt. 24, assistant in a tobacconist shop, had for several years suffered from almost continuous indigestion, severe pain and vomiting, with on one occasion hæmatemesis, which, notwithstanding treatment, had become gradually worse, until she was quite unable to retain even milk. In consequence she had become weak and emaciated. On the afternoon of March the 7th, while at work, she was suddenly seized with agonizing pain in the region of the stomach, shooting through to the back, and rapidly spreading over the whole abdomen. She was taken home in a cab and put to bed. Hot fomentations were applied, but after a night of continued severe pain she was transferred to the Infirmary the next forenoon.

On examination, her condition was very similar to the other case, but she was much more collapsed, with pinched face, dry tongue, subnormal temperature; pulse 120, respiration thoracic and 32, and pain excessive and general over the abdomen, which was rigid, tympanitic, but not much distended, liver dulness being absent. Though the general condition was bad an operation was at once proceeded with, the usual incision from the ensiform cartilage to the umbilicus being made. Gas and flaky pus escaped; the stomach was found congested; about its middle it was found to be contracted into a hard mass about an inch in diameter and an inch long, this forming an hour glass contraction separating a dilated cardiac pouch from a pyloric one. In the centre of this contraction, at the lesser curvature at the point of attachment of the gastro-hepatic omentum, was found an irregular perforation with sharp edges

large enough to admit the end of the little finger. This was surrounded with flaky lymph. The difficulty was how to close this opening without further diminishing the calibre of the communication between the pouches, this already being hardly sufficient to admit the forefinger and evidently being due to the cicatricial contraction of a long-standing gastric ulcer. The whole condition thoroughly explaining the prolonged gastric symptoms from which she had so long suffered. What he (Dr MacGillivray) did was to make a longitudinal incision in the




long axis of the stomach about 3 inches in length and having the perforation in the centre. This was then stretched vertically as in pyloroplasty and the vertical edges united with a continuous silk suture through all the coats of the stomach, a large number of superficial interrupted Lember's sutures being then inserted, the perforation being thus closed and the normal lumen of the stomach re-established at the same time. Fig. 1 shows the line of incision, and Fig. 2 the condition after suturing. The

whole abdominal cavity was found to be bathed with pus, in the subphrenic region, behind the liver and spleen, and down into Douglas' pouch. The cavity having been most thoroughly washed out with hot saline solution, a counter opening being made supra-pubically, glass drainage tubes were inserted both at the upper and lower openings; and the wounds then closed by suture.

After the operation, which lasted about an hour and a quarter, she was somewhat more collapsed, the pulse being small and compressible and about 130. After the administration of an enema of brandy and beef tea she rallied wonderfully and passed a fair night, with little pain but considerable restlessness and only snatches of sleep, nothing being given by the mouth, but the nutrient enemata being continued every four hours.

The next day, March 9th, she had rallied wonderfully, the pulse being stronger and 104, the temperature 99·2, and the respiration 20. She was very cheerful, but complained of excessive thirst. Enemata of hot water were given to relieve this, and the nutrient enemata continued. Directions were given that if there was no sickness, tea-spoonfuls of hot water were to be administered by the mouth every half hour, commencing in the evening, and these gradually to be altered to milk and hot water.

On dressing the wounds there was found a small quantity of blood-stained sero-purulent discharge in the upper dressing, but only a very small quantity of blood-stained serum in the dressing around the suprapubic opening; pain and distention of the abdomen had disappeared.

On March 10th she was quite cheerful and happy, having had a good night, and the pulse and temperature were practically normal. There had been no sickness, and she had been able to take the teaspoonfuls of milk and water by the mouth regularly. Small quantities of chicken tea in addition to the milk were ordered to be administered, but the nutrient enemata to be still continued. The lower tube was removed as there was no discharge, the upper being still left *in situ* as there was still a small quantity of serous discharge.

From that time onwards the course of the case was uneventful; the upper glass tube was removed on the fourth day, a short piece of small rubber drainage tube being sub-

stituted for a few days longer. At the same time the necessity for nutrient enemata ceased, the patient being able to take sufficient fluid nourishment by the mouth—milk, milk and raw eggs beaten up, veal and chicken tea, and gradually farinaceous articles of diet. She never complained of the least pain or indigestion, and the bowels acted regularly. No medicine was administered beyond Hæmatogen, which she took with relish, and which seemed to strengthen her. She was allowed out of bed about the third week, and rapidly gained strength and weight. Except for a small sinus at the upper wound, every thing had soundly healed, and this had closed ten days later. She was sent to the Convalescent Hospital at the end of April, well and strong with no trace of indigestion, and able to eat any simple food. When seen a few days ago, more than three months after the operation, she was in perfect health, well nourished, with good colour, and taking ordinary food with appetite and relish. This case is of special interest as being exactly similar to one reported by Mr Morse of Norwich in the *Lancet* of May 13th, 1899, with the important difference that in his case no perforation had taken place; the symptoms, pathological condition, treatment, and result were, however, precisely similar.

5. *Dr Alexander James* exhibited—

(*a*) CASE OF CIRRHOSIS OF THE LIVER. The patient was a man aged 45, and dock-labourer, and was admitted to the Infirmary, December 2nd, 1898. He had been very alcoholic and had had jaundice but now he had great ascites and some right-sided pleural effusions. The peculiarity in his case, however, was that tapping the pleural cavity emptied the abdominal. Over 400 oz. of fluid had been on several occasions drawn from his pleura and there was no doubt that there was a communication through the diaphragm. In fact it seemed that the abdominal cavity could be rather more easily emptied through the pleural than directly.

(*b*) CASE OF HEMICHOREA. The patient was a lad of 17. A mason who had been admitted to the Infirmary, April 8th, 1899.

He had had rheumatic pains for some months and chorea limited to the right arm, leg and right side of face for three weeks. Over those parts sensibility was slightly impaired and the deep reflexes of the right leg and right arm were slightly

increased. The interesting point was that ten years previously he had had an accident to his right foot, necessitating the amputation of the right great toe. For the development of the chorea in the right side, this was believed to be the determining cause.

6. *Dr Allan Jamieson* exhibited—

(*a*) A CASE OF TROPHO-NEUROTIC LEPROSY IN AN EARLY STAGE.

B. D., aged 8, born in British Guiana. A well-grown, stout boy, clever and active. No hereditary history of leprosy in his family. Father a tall, strong, dark Creole, mother fair. Six years ago he occupied the same bed as a nurse, an apparently healthy woman, but who was discovered to be suffering from an ulcer of the leg which was pronounced to be leprous by an experienced doctor. She was sent to Tobago. It is believed that the ulcer healed, but her subsequent history cannot be ascertained. Two years since, blotches appeared on his face and on the lower part of his back ; at first these were so faint that they were only perceptible to his mother who anxiously watched him. One also shewed itself on the right ear, but this is no longer visible. There were no distinct prodromal symptoms, indeed, with the exception of occasional slight feverish attacks he has, and does enjoy excellent health. There are now some faintly purplish blotches the diameter of a pea on chin, and some larger still paler ones on cheeks scarcely, if at all, elevated above the surface. There are none on the eyebrows or forehead. The nose is broad, the hair dry and somewhat thin. On the forearms, especially on the ulnar side, there are a number of blotches similar to those on the chin. These are rather a brownish tint when the surface is first uncovered, but on exposure assume a more purplish hue. On the upper arm the marks are more pigmented stains. On the legs, particularly behind, there are blotches of the same character. On the gluteal region on the right side there is a patch closely resembling morphœa, or localised scleroderma. It is the size of a crown piece, is a very pale lilac white in centre, surrounded by a slightly darker violet ring. On the back are a very few smooth brown stains, some enclosing a white spot, or a white spot without any pigment. The white spots are scarcely as large as a pea. Tested for anæsthesia

or hyperæsthesia the white spots are a little less sensitive than the neighbouring skin, but not definitely anæsthetic. The ulnar nerve is not thickened, nor can any peripheral nerve alterations be discovered. The hands are large, and the joints are swollen, and in buttoning and unbuttoning his clothes he seems to use his fingers with less nimbleness than he should. But he grasps firmly, and does not drop articles he holds. The case exhibits a very early period of macular leprosy.

(*b*) Two cases exhibiting the contrast between LUPUS VULGARIS as affecting the face, and SYPHILIS probably inherited, attacking the same region.

Both cases were women aged about thirty. The disease in both commenced about the age of nine. In the instance of lupus the disease was still progressive and fairly symmetrical. The cheeks were scarred, and the cartilages of the nose had been destroyed though now wholly cicatrised. There were groups of lupus nodules at the sides of the cheeks and on the neck. The mouth was narrowed by the cicatrices so that cracks readily formed at the angles, and there was an ulcer under the upper lip. In the specific case there had been a like destruction of the nasal cartilages, and of a portion of the septum, and there was a muco-purulent discharge. The interior of the nose had been examined by Dr M'Bride, who found lesions which he regarded as specific. The mouth was diminished in size as in the other case. The resemblance between the two cases when looked at was remarkable, but the cicatrices in the specific case were smoother than in the instance of lupus, and the only place in which there was any active disease was just below the nose where there was an ulcer, now rapidly healing under iodide of potassium. Whether the syphilis was inherited or acquired there was no history to guide. It was probably due to inherited disease, however, and an example in that view of late hereditary manifestation.

## II. Exhibition of Specimens

1. *Dr W. Russell* exhibited—

(*a*) Two specimens illustrating one of the unusual positions of the VERMIFORM APPENDIX. In the first, the appendix was as long as a finger; it lay behind the cæcum and ran up the ascending colon. In the second specimen the appendix lay

behind the ascending colon, the tip of it being gangrenous. In neither case would Mr Burney's point have been of any value, and indeed would have been worse than useless.

(*b*) Specimens from two cases of PERITYPHLITIS not originating in the appendix. In the first case, there was a history of fœcal impaction, and while the appendix was found healthy, fœcal ulcers were present in the cæcum, one of which had perforated. In the other case which was operated on the appendix was unaffected, but the caput cæcum was inflamed, and this was found to have been caused by fœcal ulcers in the cæcum.

2. *Dr R. A. Fleming* and *Dr Welsh* exhibited a series of pathological specimens, illustrating tubercular lesions in different organs, including peribronchial tuberculosis in the lung of a boy aged 15 years, showing a very curiously limited spread of the disease; tubercular pericarditis which, as Dr Fleming remarked, was of rare occurrence; tubercular ulceration of the intestine; tubercular disease of the larynx; tubercular meningitis of brain and spinal cord and one specimen of a tubercular nodule in the pons; tubercular disease of the suprarenals from three cases of Addison's Disease with a couple of tubercular suprarenals which had not been associated with well-marked pigmentation.

Dr Welsh exhibited a series of lungs, illustrating the formation of cavities in pulmonary tuberculosis.

3. *Mr F. M. Caird* exhibited—

(*a*) Drawings and preparations illustrating large multiple pedunculated mesenteric cysts which contained chyle and serum removed from the abdomen of a child whose symptoms resembled those of tubercular peritonitis. Recovery was uninterrupted.

(*b*) A map of cicatrix and subcutaneous tissue containing a small nodule of carcinoma recently removed from the breast of a patient on whom excision of the mamma had been performed nine years ago. There had been recurrence in the shape of solitary isolated small nodules in the pectoral region at intervals of four, six, and seven years. On the last occasion the whole cicatrix had been cleared away. The patient all along has been gaining in weight and is 68 years of age.

(*c*) A large cystic tumour of the testicle.

(*d*) Malignant degeneration of a uterine fibroid removed by panhysterectomy after the method of Kelly. After removal there was found peritoneal infection of the intestine in the pouch of Douglas, but the patient still progresses favourably.

4. *Mr C. W. Cathcart* shewed a specimen of MULTIPLE GASTRIC ULCER. The patient had been a young woman in domestic service in the country. The symptoms pointing to rupture had first appeared seven days before her admission into the Infirmary, but the case had been taken for one of appendicitis. When she came to Edinburgh peritonitis was far advanced. The perforation in the anterior wall of the stomach was sewn up and the peritoneum and pelvis washed out so far as her strength would allow. She died, however, about ten hours after the operation, and her death was much hastened by persistent hæmatemesis. This was found at the *post-mortem* examination to have been due to a second ulcer which had no doubt been damaged by the handling of the stomach during the operation. A third ulcer was also found. It lay on the posterior wall and over an area of about the size of a threepenny piece, its base was formed merely by a thin layer of peritoneum. Thus, even had the first, or ruptured ulcer, been taken in time, the complications arising from the other ulcers would probably have shewn themselves before long.

## Meeting IX.—June 7, 1899

DR WILLIAM CRAIG *in the Chair*

### I. EXHIBITION OF PATIENTS

1. *Mr Shaw M'Laren* exhibited a patient after amputation at the hip-joint for injury. Boy, age 14, had his thigh crushed by machinery. Recovery after amputation at hip for disease occurred with some frequency, but recovery after amputation at hip for injury sufficiently infrequent to justify exhibition of patient. Other points of interest. (*a*) Hæmorrhage was controlled with ease and complete success by digital pressure on aorta; this might often not be so easy on an adult or fat patient. (*b*) Patient's pulse less after operation, though no

appreciable blood lost; transfusion of a pint of hot salt water through mediary cardia induced striking improvement. (*c*) In comparative collapse, which continued, no alcohol was given, but hot strong coffee. (*d*) One night—twelve hours—elapsed between accident and patient's arrival at hospital. Possibly recovery aided by this; possibly "primary" amputations should be delayed till shock of accident well over, so long as septicæmia can be prevented.

2. *Mr C. W. Cathcart* exhibited a patient who had recovered after an operation for RUPTURED VERMIFORM APPENDIX WITH GENERAL PERITONITIS.

Dora S., æt. 26, was admitted to the side room of Ward V. on February 2nd with symptoms of general peritonitis—pulse 126, temperature 102°.

On January 29th she had felt shivery all afternoon, but had slept well. On the 30th she had abdominal pain which she thought was due to a "sore stomach," and she passed a restless night. On the 31st the pain was very severe. She went to have a dress fitted on however, but fainted during the process. On February 1st the pain became localised in the region of the appendix; her medical attendant was called in and relieved her pain with opium. As she did not improve, she was sent into the Infirmary on the 2nd of February.

An operation was performed without delay. A vertical incision was made through the right rectus muscle, and the abdominal cavity opened. The vermiform appendix hung over into the pelvis, and a large sloughy ulcer occupied one side of its point of junction with the cæcum. Putrid pus lay round the ulcer and in the pelvis without any encapsulation. The small intestines in the pelvis and lower part of the abdomen were matted together with recent lymph, and as the adhesions were freed, pockets of purulent fluid were opened up.

The distal part of the appendix was removed beyond the ulcerated spot. The distended small intestines were then drawn out of the wound, the adhesions freed, and the surface freely douched with hot saline solution ($\frac{3}{4}\%$). After about $\frac{2}{3}$ of the small intestine had been thus dealt with, the subsequent coils were found to be unaffected, and were not disturbed. The cæcum had to be punctured and emptied of gas before the intestines could be returned, the aperture was stitched up with

Lembert's stitches. A glass drainage tube was put into the pelvis, and the region of the sloughy ulcer at the root of the appendix was packed round with gauze, which was left in the wound. For some days the patient's condition was critical, but she gradually improved and is now in very good health. A great deal of sloughing of the rectus muscle occurred, but there was no return of active peritonitis. The wound has now healed firmly except at one spot where there is a small fistulous opening into the bowel. The opening however is valvular, for neither gas nor fœculent matter escape outwards, while fluids injected into it are discharged per rectum.

The patient's recovery seemed to be due to the adoption of the method of freely pulling the intestines out of the abdomen, so as to cleanse and douche them more thoroughly than would have been otherwise possible.

*Mr F. M. Caird* exhibited a case to illustrate a fresh attack of SYPHILIS in a patient who had suffered from the congenital form of the affection.

J. B., æt. 24, a well-developed and otherwise healthy male, has the characteristic saddle nose and beetle brow of hereditary syphilis, and volunteers the statement that in early infancy he suffered from snuffles. There is no other certain evidence of the disease.

About two months ago he came to hospital with a characteristic hard sore on the frenum, and this was followed by a characteristic roseolar and papular rash with affection of the mouth.

## II. EXHIBITION OF PATHOLOGICAL SPECIMENS

*Dr T. Shennan* exhibited—

(*a*) MALIGNANT PUSTULE with microscopic preparation, showing the bacilli anthracis in the minute vesicles surrounding the necrosed central part.

(*b*) SARCOMA OF CEREBRUM in tip of temporal lobe.

(*c*) TESTICLE AND EPIDIDYMIS, showing in the epididymis a cyst surrounded by thick calcareous walls—probably surrounding an old tuberculosis.

Continuous with this deposit the tunics of the testicle are also calcified except a small part in front. Testicle appeared to be unaffected. From a man æt. seventy-five years.

(*d*) INFARCTS OF KIDNEY. The cortex is practically filled with infarcts, with congested parts between containing much blood pigment. From a case of uræmia.

(*e*) TUBERCULOUS NODULE IN WALL OF HEART. Invasion in this case had taken place by way of the cervical and tracheal glands and thence to glands along aorta.

(*f*) PULMONARY TUBERCULOSIS IN DIABETES. The lungs show nodules of varying size undergoing very rapid caseation and softening.

(*g*) A SUPERFICIAL ULCER from the same case, with dry glazed slough on surface.

## III. ORIGINAL COMMUNICATION

## A CASE OF HEREDITARY SYPHILITIC OSTEITIS

By ALEXANDER JAMES, M.D., F.R.C.P.Ed., Physician to the Royal Infirmary, Edinburgh

J. W., aged 18, a worker in bottle works, was admitted to the Royal Infirmary, December 8, 1898, complaining of swelling in the right leg, headache, and weakness in the right hand and arm.

*Family history.*—Although syphilis is denied on the part of either parent, there is practical certainty of its existence. The father is aged 40, the mother 47, and the following is the list of children and miscarriages.

1. A boy, by another father. Had pains and swellings in his legs, and the pains were distinctly worse at night. Died, aged 13 years, from inflammation of the lungs.

2. Boy, died aged 7 weeks, cause unknown.

3. Boy, died aged 6 days, had a swelling on his back at birth—[spinabifida (?)]. Mother says that she hurt her back when pregnant with this child.

4. The patient.

5. A miscarriage at the 3rd month.

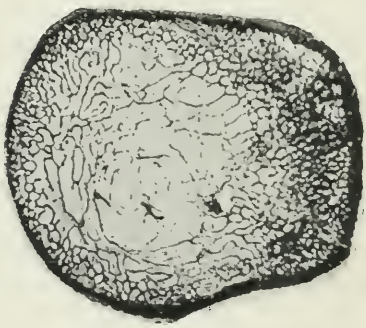
6. Boy, died aged 11 months, cause not clear, but had a rash on the skin.

7 and 8. Two miscarriages, about the 3rd month.

9, 10, 11 and 12. Four children, now alive and healthy.

This patient had no snuffles nor rash in infancy, but had keratitis, leaving spots on the eyes, when aged 2 years. At the age of 3 had scarlatina, and at this time a slight squint of

AGED 10 YEARS.



TRANSVERSE SECTION OF RIGHT TIBIA.

TO ILLUSTRATE DR JAMES' PAPER.

AGED 18 YEARS.

TO ILLUSTRATE DR JAMES' PAPER.

the right eye was noticed. At the age of 8 had measles and whooping-cough. Never had any accidents.

His present illness seems to have begun when he was about 9 years old, shortly after his attack of whooping-cough. He began to complain of pains in his arms and legs, the pains being much worse at night. About that time (1891) Dr John Thomson saw him and found the lower ends of both humeri thickened and tender, and a large flat node on the right tibia, about the junction of the middle and upper third. Dr Thomson also found a similar but smaller node on the left tibia, and a thickening of the spine of the right scapula. Iodide of potass was administered, and the patient improved very much.

In 1893 Dr Thomson again saw him, and found nodes on both tibiæ, especially the right, and a thickening at the lower end of the right humerus. The first photograph, for which I have to thank Dr Thomson, shows his condition at that time.

In 1897 he came into my ward in the Infirmary, complaining of pains in the legs, especially the right. At that time I found diffused thickening and elongation of the right tibia. The left tibia was also enlarged, but not so much so. No complaint was made then as regards the arms. He improved very much under the iodide of potass and rest, and after some weeks was discharged much improved.

In May 1898, he noticed that the index and middle fingers of his right hand were becoming weak. Gradually the other fingers became involved, so that at the end of a month, movements of the fingers and hand were lost. About the end of October he began to suffer from headaches. These were worse at night, and the pain seemed always to be most severe over the left eyebrow. About a week after this, twitchings of the muscles of the right hand and arm, occurring with periods of unconsciousness, were noticed. About the same time vomiting occurred at times. These symptoms led him again to seek admission to the Infirmary, and he was admitted as above.

*State on admission.*—He is a badly-grown boy, of about 5 feet 4 inches in height, and weighs 6 stones. His muscularity is very poor, he is apathetic, his pulse is usually about 70, and his temperature normal (see photograph No. 2).

*Locomotory system.*—The right tibia is thickened along its whole length. It is slightly curved inwards, and is about ¾ inch longer than its fellow. The left tibia seems also somewhat enlarged, but very slightly so in comparison with the right.

*Nervous system.*—Sensibility to touch, pain and temperature appears unimpaired all over. It is possible there may be some impaired sensibility in the right arm, but the patient is so apathetic that we cannot make sure of this. He states that of late his vision has become somewhat blurred, and he notices this especially on awaking from sleep in the mornings. On examination of the eyes, the appearance of slight old keratitis was found on the right. Both pupils reacted normally to light and accommodation. There was a slight amount of optic neuritis, especially on the left eye.

Hearing and taste were apparently normal, and he stated that he never possessed the sense of smell.

Voluntary motor power in the muscles of the right hand and forearm was practically lost. As regards reflexes, the plantar and epigastric were rather more marked on the left side; the cremaster was present on both sides. The knee and ankle jerks were absent on both legs.

*Hæmopoietic and circulatory systems.* — The lymphatic glands on both groins were enlarged, as also were those in the neck and submaxillary regions, and the epitrochlear glands. Those in the axillæ were not markedly enlarged. The spleen was enlarged, measuring 7½ by 5 inches. The thyroid was normal. The heart appeared normal as regards size, and its sounds were closed. The pulse, about 70 per minute, was of low tension, and the arterial walls somewhat thickened. The thickening was well marked in the veins of the legs; the right long saphenous vein could be traced, like a fibrous cord, from the dorsum of the foot to the groin.

The respiratory system was normal.

The urine, normal in amount, was of spg. 1015, neutral in reaction, and showed usually a deposit of amorphous phosphates.

The diagnosis made was hereditary syphilis—nodes followed by chronic osteitis, specially of the right tibia, producing a condition of local gigantism—tumour, probably gummatous, about the arm area on the left side of the brain.

The patient was put on large doses of iodide of potass, and he improved somewhat as regards the headaches and general condition. After two weeks in hospital, he determined to go home. There his condition soon became worse, and he died comatose on January 18th. The *post-mortem* examination was made, under rather unfavourable conditions, at his own house. A gummatous tumour about the size of a golf ball was found about the middle of the left rolandic area. The nerves were not examined, but a disc-like portion of the enlarged right tibia, near its upper part, was obtained. This showed great enlargement, measuring in diameter $1\frac{3}{4}$ inch by $1\frac{7}{16}$ inch. It was softened in corrosive and cut. Under a low power, as the photograph shows, the section appears much like a section of a cancellated bone. It shows a thin rim of compact tissue around, with cancellated tissue all through, and practically no medullary cavity. With higher powers the interspaces of the trabeculæ are seen to be filled up mainly by fat cells. Here and there, however, a few marrow cells are to be seen.

*Remarks.*—Here we believe we have a case of a distinct specific lesion, viz., the formation of a node, followed by the disappearance of the node and the development of a less specific condition, an osteitis. In the development of the node we recognise what Fournier[1] and others would call syphilis "en nature." In the osteitis we recognise the existence of pathological characters which would be called "parasyphilitique," "dystrophique," or "toxinique." In this latter condition we recognise as the factor the innate constitutional deficiency, rather than the specific virus.

A point worthy of remark here is that this patient, after getting quit of the nodes on the bones and showing only the osteitis, yet died eventually of a gumma of the brain.

This osteitis has, as the section of the bone shows, caused, as regards its tissue, an increase in quantity at the expense of quality, a sacrifice, as it were, of development to growth. The affected bone, composed mainly of cancellated tissue with no medullary cavity, has, as it were, reverted to an earlier developmental stage.

With the increase in thickness there has also been an increase in the length of the bone, a partial gigantism. Similar cases have been described by Fournier and others, and it has

[1] Stigmates dystrophiques de l'hérédo-syphilis. Edmond Fournier. Paris, 1898.

been asserted that, as the result of hereditary syphilis, general gigantism may be brought about. Fournier gives several examples of publicly exhibited giants who were undoubtedly of syphilitic heredity. He also points out that dwarfishness can, on the other hand, be attributed often to hereditary syphilis, an illustration of what we often see in nature, viz., the same cause producing opposite results in different cases.

A further point of interest here is, as regards how the elongation of the bone has been brought about. As we all know, a bone is said to elongate by the formation of new bone and cartilage between the shaft and epiphyses. Certainly in our patient, dying at the age of eighteen, there was time left for elongation to take place at those points. But the microscopic examination of the shaft suggests that elongation must have taken place in it also. The same pathological change which led to an increase in the thickness of the shaft, with diminution in the size of the medullary cavity, must certainly also have led to its elongation.

Finally, whilst the usual effect of syphilitic or parasyphilitic constitutional conditions is, as regards bone, to cause an undue condensation rather than a replacement of its compact by cancellated tissue as in this case, it may be suggested that here we view the pathological process at one stage only. Had this boy lived a few years longer, we might have found this "rarified" bone becoming again compact bone, just as it had done at an earlier developmental stage. That is to say, we might have found development again supervening on growth. It is evident that in this case the amount of compact tissue would have been excessive.

## Meeting X.—July 5, 1899

SIR JOHN BATTY TUKE, *President, in the Chair*

### I. ELECTION OF MEMBER

Robert Black Purves, M.B., C.M., F.R.C.S.Ed., 17 Walker Street, Edinburgh, was elected an Ordinary Member of the Society.

### II. EXHIBITION OF PATIENTS

1. *Mr H. J. Stiles* exhibited—

(*a*) Child, two months after operation for large HYDREN-

CEPHALOCELE, with stereoscopic photographs before and after operation, by Dr A. Watt.

The child, now aged eleven months, was shown to the Society four months ago.[1] The large size of the tumour, and the deformity produced by it, is well shown in the photograph by Dr Watt. Simple tapping with fine trochar and canula was employed on two occasions, fifteen ounces of clear fluid being drawn off on the first occasion, and eighteen ounces on the second. The fluid, however, rapidly reaccumulated.

On April 28th the following operation was performed. After slowly drawing off as much of the fluid as possible (twenty ounces), an incision was made into the sac in the frontal region, when about four ounces more fluid escaped. On introducing the finger, the anterior part of the sac was found to be superficial to the frontal bone, which was well developed. Behind the frontal bone the finger passed through a constriction in the sac into a larger posterior compartment into which the posterior part of the cerebral hemispheres herniated through a large deficiency in the cranial vault. A second incision was made into the sac in the occipital region; on introducing the finger it was found to occupy the subarachnoid space, and could be made to touch a second finger introduced into the anterior opening. The supra-occipital bone had not developed, and the parietals here represented only by the part corresponding to the anterior inferior angle. Two large elliptical portions of the sac-wall were then removed, the one in front and the other behind the constriction. The edges of the wound were then brought together over the protruding hemispheres by interrupted sutures of fishing gut, while the skin edges were more accurately united by a continuous suture of horse-hair; no drainage. The child did not suffer so greatly from shock as one would have expected. The temperature rose from one to two degrees for the first few days. The wound was dressed for the first time on the seventh day, when it was found that there was slight suppuration, which appeared to be confined to the superficial part of the wound. On removing the stitches clear cerebro-spinal fluid issued from the openings. Two days later the wound was looking well, but a considerable quantity of clear fluid escaped from the anterior

[1] See page 133.

incision ; the fluid continued to drain away for a month, after which the wound completely closed throughout. Up to the present there has been no reaccumulation of fluid. Instead of the lobulated sac which, before operation, was larger than the rest of the child's head, there is now a tumour, the size of an orange, projecting through the deficiency in the cranial vault. The tumour consists of the posterior part of the cerebral hemispheres. The condition was that of a large hydrencephalocele which had ruptured anteriorly, so that the fluid in the anterior part of the sac was situated between the scalp and the pericranium covering the frontal bone.

(*b*) Child after operation for EXTENSIVE TUBERCULOUS OSTEOMYELITIS OF TIBIA, with skiagram by Dr Rainy.

The child, two and a half years old, was admitted into the Children's Hospital with a tuberculous abscess over the inner side of the lower part of the right tibia. The tibia, in its lower half, was nearly twice the normal thickness ; the thickening faded off towards the upper epiphysis. The skiagram shows that the thickening is mainly due to the formation of a sheath of new bone between the thickened periosteum and the original compact bone of the shaft. The medullary canal is excavated throughout and occupied by cheesy and tubercular granulations which have given rise to the lighter shadow corresponding to the enlarged medullary canal. The operation consisted in laying open the whole length of the medullary canal by chiseling away the inner surface of the shaft, after turning aside the periosteum. The medullary canal having been thoroughly cleaned out with a sharp spoon, and sterilised iodoform rubbed in, the wound in the soft parts was completely closed. The cavity filled with blood clot which became organised. The wound was dressed on the eighth day to remove the stitches ; a small quantity of yellow serum was let out with sinus forceps. At the next dressing the wound was firmly healed.

The pathology of this condition is identical with that of tuberculous dactylitis : the subperiosteal new bone is not tubercular ; it is the result of the irritation produced by the tubercle in the medulla. Merely opening the abscess with scraping of the bone in its neighbourhood can not be expected to bring about the cure of such a condition. The proper treatment is to lay open the whole medullary canal in the manner above indicated.

(*c*) Child after excision of wrist for TUBERCULOUS DISEASE, with skiagrams by Dr Rainy, showing the result of treatment and a comparison with the opposite wrist.

2. *Dr Alexander Bruce* exhibited a patient suffering from AUDITORY APHASIA. After a sudden seizure in March there was a degree of paralysis of the right arm and an immediate loss of power of comprehension of spoken words. The patient began to speak without interruption but her speech was quite unintelligible. She lost the power of reading and, even after the right hand had recovered from its paralysis, of writing. A demonstration was given of the alteration of the patient's speech.

## III. Exhibition of Pathological Specimens

1. *Dr Alexander Bruce* exhibited SECTIONS OF THE SPINAL CHORD from a case of pure progressive muscular atrophy, and from one of amyotrophic lateral sclerosis. It is the opinion of many neurologists of authority that in progressive muscular atrophy there is invariably a degeneration of the crossed pyramidal tracts in the cord. The sections derived from the case of progressive muscular atrophy, which had been carefully observed clinically, showed that the crossed pyramidal tracts were quite normal, and in marked contrast to those from the amyotrophic lateral sclerosis where they were markedly degenerated.

2. *Mr H. J. Stiles* and *Dr Harry Rainy* exhibited—

(*a*) Skiagram of TUBERCULOUS ANKLE showing cartilages of the joint replaced by granulation tissue. At the operation it was found that there was much more disease in the neighbourhood of the lower epiphysis of the tibia than the skiagram would lead one to anticipate.

(*b*) Skiagram showing separation of LOWER EPIPHYSIS OF HUMERUS, the result of a fall upon the flexed elbow. The photographs bring out very forcibly the upward and backward displacement of the epiphysis, along with the bones of the forearm, and they show how readily such an injury might be mistaken for a posterior dislocation of the elbow. With the object of ascertaining the best position in which to treat the

fracture, skiagrams were taken, first with the elbow flexed as far as possible, and then with the joint straightened ; the former position gave the best apposition, whereas the latter position increased the displacement.

(*c*) Skiagram showing FRACTURE OF FEMUR (five weeks after the injury) at junction of upper and middle third of shaft. There was nearly an inch of shortening. The skiagram shows that the upper fragment is flexed, abducted and rotated outwards, while the lower fragment is drawn upwards, and lies behind and to the inner side of the upper fragment. The lower end of the upper fragment could be very distinctly felt under the anterior and outer aspect of the thigh.

3. *Dr Harvey Littlejohn* exhibited specimens from—

(*a*) A case of STRANGULATION OF THE BOWEL. About two feet of the ileum had passed through a hole in the great omentum, and was in an almost gangrenous condition. The patient, a printer, was suddenly seized with pain in the abdomen and vomiting, which lasted intermittently until death occurred at the end of forty-eight hours. He continued at his work until a few hours before death.

(*b*) A case of INTUSSUSCEPTION, which occurred in a woman aged thirty-five. The symptoms came on during the night, after a supper of tinned sardines, and they were at first attributed to poisoning from this cause. Death occurred within forty-eight hours. *Post-mortem* an intussusception of the ileum just above the cæcum was found ; there were comparatively slight signs of peritonitis, but great œdema of the contained end from constriction of the neck.

(*c*) The TONGUE, FAUCES, ŒSOPHAGUS, STOMACH, AND INTESTINES from a case of hydrochloric acid poisoning. The man took two ounces of commercial spirit of salt suicidally, and was at once seized with violent vomiting; he died in an hour and a quarter. The whole alimentary tract down as far as six feet below the duodenum showed corrosion of the mucous membrane, with blackening of the surface of the stomach and upper portion of intestine. There was no perforation.

(*d*) The STOMACH of a man who took suicidally half a wineglassful of chloride of zinc ("soldering fluid"). He was found lying on the floor, vomiting yellowish-green material and purging. He complained of great pain in the abdomen, and was removed to the Royal Infirmary, where he died with

symptoms of enteritis on the seventh day after taking the poison. The stomach was blackened, the mucous membrane being corroded, and the coats of the stomach softened. It presented an appearance similar to that of the case of HCl poisoning, the corrosion, however, being less intense.

4. *Mr J. M. Cotterill* described a case of RUPTURED OVARIAN DERMOID CYST which he had removed a few days ago from a woman aged 34. She had had a child three months previously, the labour being satisfactorily got over, though she was only seven months' pregnant.

After the birth of the child a lump had been noticed in the pelvis about the size of the fist. This had given her little trouble till two days before admission to hospital, when she was suddenly seized with an agonising pain across the lower part of the abdomen.

She was dosed with opium for two days, and then sent into hospital as a case of appendicitis. On admission it was evident she was suffering from general peritonitis, and the diagnosis was made as probably one of purulent salpingitis. The abdomen was opened, the diagnosis of general peritonitis confirmed, and the contents of a ruptured cyst were found in large quantity free in the abdomen. Lying low down in Douglas' pouch was found a ruptured dermoid, the size of a cocoanut, containing a large mass of hair. The cyst, together with the right ovary and tube were quickly removed, and the abdomen washed out, a drain being left in. She got over the operation well, and promised well for some time, but the peritonitis had not been caught early enough, and it proved ultimately fatal.

## IV. Original Communications

### 1. A CASE OF CEREBELLAR TUMOUR, CONSIDERED WITH REFERENCE TO ITS LOCALISATION

By ALEXANDER BRUCE, M.A., M.D., F.R.C.P.Ed., F.R.S.E., Assistant Physician to the Royal Infirmary, Edinburgh. Surgical part by J. M. COTTERILL, M.B., F.R.C.S.Ed., Surgeon, Royal Infirmary, Edinburgh

AT the January meeting of this Society I drew attention to the importance of a knowledge of the relation of the main conducting strands of the cerebellum for the interpretation of the results of experimental lesions on that organ, and stated that this know-

ledge might be of practical use to the clinician in elucidating the symptoms of cerebellar tumours, and in determining their localisation. This communication has been published *in extenso* in the *British Medical Journal* of May 6. Since that time I have been on the outlook for cases by which the views then expressed might be put to the test. During the month of April a patient came to the medical waiting-room presenting the symptoms of cerebellar tumour. His condition, on examination, afforded such an excellent illustration of the truth of the principles laid down in my communication, that it seems worthy of being brought under your notice to-night. Mr Cotterill will describe to you the difficulties met with in the surgical procedures undertaken with the view of removing the tumour, and will indicate his opinion as to the part which surgery may hope to play in the future in dealing with cerebellar disease.

The history of the case was briefly as follows :—

W. K., aged thirty-four, married, hairdresser by occupation, was admitted to Ward XXII., Royal Infirmary, on April 3rd, 1899.

His family history was unimportant. He was, and had all his life, been a total abstainer, and he had never suffered from any form of venereal disease.

Five years ago without assignable cause the hearing of his left ear suddenly became impaired. In three or four days the deafness in this ear became absolute, and has remained so ever since. He had no other symptoms until two years ago, when he began to find that he could not walk in the dark or ride his cycle, because he felt as if he were going to fall to the left side. He states that he could always feel the ground quite distinctly while walking. At this time he for the first time noticed a symptom, which persisted to the end, and which appears to be of considerable importance for a proper understanding of the case. He found that, in order to execute any movement with the left arm or leg, it was necessary to make a special effort of will or to direct his attention closely to the limb, until the movement was completed. If he did not do so the desired movement was very irregularly performed. His gait became increasingly unsteady, and was sometimes attributed to intoxication. At one time, for a week or so, his face was twisted to one side. He does not know to which, but in all probability it was to the left side. Seven months ago he had two or three attacks of

vomiting, which could not be ascribed to any cause. He remained quite free from such attacks till the 3rd of May, when there was a slight recurrence. Six months ago he began to suffer from severe, left-sided occipital headache. About the same time he began to have attacks of dimness of vision, which would supervene suddenly, as if a mantle had fallen in front of his eyes. During the last two or three months the dimness of vision has become more continuous, and has progressively increased, especially in the right eye. He also complains of a dull pain in the small of his back. This pain becomes more severe towards the end of the day.

On examination, he is a well-developed man, and shows no very obvious morbid appearances, beyond a certain look of depression. His temperature since admission to hospital has been persistently slightly subnormal, averaging 97·4, except on one occasion when there was a slight febrile attack, which was probably due to a chill.

The examination of the alimentary, circulatory, respiratory, urinary, and integumentary systems gave negative or unimportant results, which need not be quoted here, as they have no bearing on the case.

*Nervous system.*—A continuous gnawing pain in the lower part of the back, worse when patient is tired, is frequently complained of. There is also severe left-sided occipital and parietal headache, with tenderness in tapping that side of the skull.

The sensibility to touch, temperature and pain were normal throughout, except on the conjunctiva of the left eye, which was anæsthetic.

His muscular sense as tested by weights was quite normal. When his eyes are shut he can accurately locate the position of his limbs with his right arm, but not with his left.

His sense of smell is unimpaired in both nostrils.

*Hearing.*—On the left side Dr M'Bride reports nerve deafness, with almost complete loss of hearing of the tuning fork, and complete loss of perception of high tones.

*Taste.*—This appears to be normal on the right side of the tongue, but is greatly impaired on the left side, especially in the anterior two-thirds. It was extremely difficult to form an opinion as to the amount of sensibility on the posterior third. Repeated examinations seemed to indicate its absence.

*Optic nerves.*—Dr Mackay reports a certain amount of

woolliness of both discs, with paleness of their substance. The right disc is more atrophied than the left. The veins are rather distended, and the arteries narrowed. The condition suggests moderate papillitis which has subsided into atrophy, more complete at present in the right eye than in the left. Refraction nearly emmetropic; a trace of hypermetropia in each eye. There are slight nystagmic movements in all positions, increased on extreme lateral movements. *On looking to the right there are rapid minute oscillations. When the eyes are directed to the left the oscillations are slower and larger.* Oscillations of intermediate extent and rapidity occur when looking upwards, and to a less extent when looking down. Convergence is moderately well performed and maintained. The field of vision in the right eye is very greatly contracted, that in the left eye is lost in the temporal side, and greatly reduced in the nasal. There is a suspicion of drooping of the right upper lid. The right pupil is larger than the left, and reacts to light rather more feebly than the latter. Both react promptly to convergence.

On looking upwards rapidly the right eyelid rises too far. There has been diplopia for fourteen days.

There is a degree of paresis of the left side of the face, the left palpebral fissure being slightly wider than the right, and the retraction of the angle of the mouth, when the attempt is made to show the teeth, is less on the left than on the right side.

The tongue is protruded in the normal manner, but the patient says that it is not so easy to turn it to the left as to the right. The grasp of the two hands is equal and fairly powerful on dynamometric examination. When both hands are held out there is a coarse tremor in the left, and a fine vibratile tremor in the right. When patient attempts to touch the tip of the nose, he succeeds perfectly with the right forefinger, but with the *left there is a coarse oscillatory movement* somewhat similar to that seen in insular sclerosis, which *causes the finger at first to miss the point aimed at, and, even after this is touched, to continue its oscillations for a short time.* The contrast is very marked when the attempt is made with both forefingers at the same time. When the two arms are held out in front of the body a fine vibratile tremor is seen in the right hand, and coarser oscillations in the left. In walking, the legs are kept somewhat widely apart, the left foot is directed straight forwards, has a slight stamping action, while the right

foot is turned outwards slightly, and is moved normally. Every few yards there is a decided tendency to stagger or lurch to the left, but never to fall to the right. The tendency to stagger to the left is greatly increased when he attempts to walk along a straight line. In this case he would actually fall if unsupported. There is no Romberg symptom. The knee jerks are present on both sides; they are not exaggerated, except perhaps very slightly on the right side. There is no ankle clonus. The plantar, abdominal, and epigastric reflexes are slightly exaggerated. The organic reflexes are normal.

From a consideration of the general symptoms of this case, namely, the headache, vomiting, staggering gait, and optic neuritis, there could be little doubt that they were due to the presence of a tumour in the posterior fossa of the skull, if not within the cerebellum itself. It remained, therefore, by a further analysis of the other symptoms to endeavour to determine the exact site of the tumour.

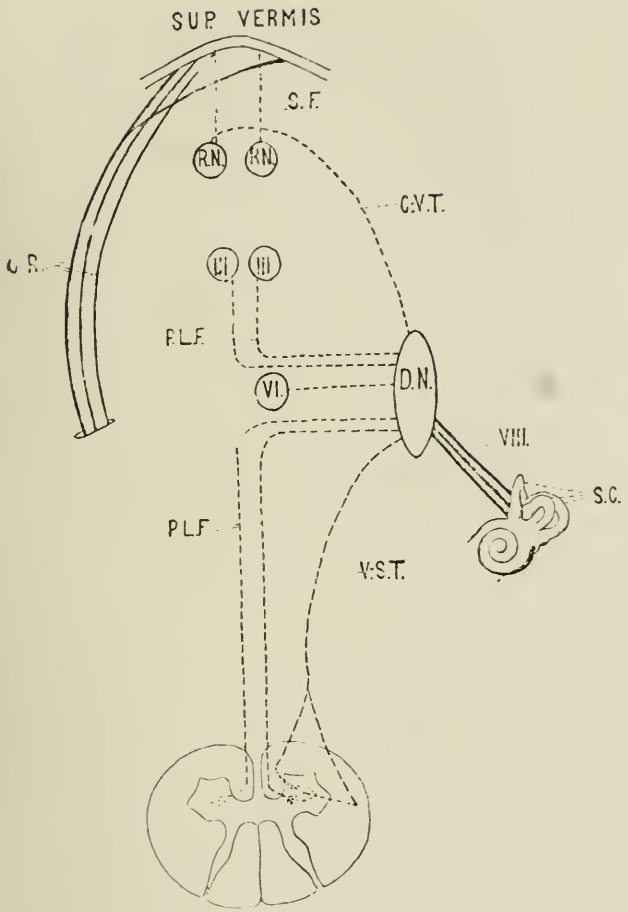
In order to explain these symptoms, I may be permitted to refer briefly to my earlier communication. The main afferent and efferent tracts that appear to be specially concerned in cerebellar coordination are in intimate relation to the middle lobe. (I except here the middle peduncles which terminate in the cortex of the lateral lobes, because their function does not appear to me to be satisfactorily determined; and confine myself to the upper and lower peduncles.) The afferent fibres derived from the spinal cord, whether directly through the direct cerebellar tract and ascending antero-lateral tract of Gowers, or indirectly from the nuclei (of the two divisions) of the posterior columns, all pass to the cortex of the middle lobe of the cerebellum. From thence other fibres pass forwards to nuclei in the roof of the fourth ventricle—the roof nuclei. From each roof nucleus a large well developed tract—the cerebello-vestibular tract—passes, as was first shown by Ferrier and Turner, towards the outer acustic nucleus or nucleus of Deiters, in which it ends. This latter nucleus also receives fibres from the semicircular canals through the vestibular root of the auditory nerve. Further, and this point appears to me to be of special importance for the comprehension of cerebellar action, the nucleus of Deiters gives origin to fibres which were first discovered by myself, and which have been since more fully traced by Ferrier and Turner, Russel, Thomas, to the

nucleus of the sixth nerve, on the same side, to the third nuclei on both sides, and to the anterior cornu of the same side of the cord, and in small measure to that of the opposite side. Each nucleus of Deiters when stimulated either from the cerebellum or the semicircular canals may thus be expected to influence the movements of the same side of the body and the lateral deviation of the eyeballs to the same side; and in smaller measure the movements of the opposite side of the body. The accompanying plate will explain these somewhat complicated connections.

About the nature of this influence there is still much discussion among physiologists into which it is not necessary to enter here. The most probable view appears to me to be that it supplements the influence of the cerebral motor cortex in giving force to the muscle called into action in any co-ordinated movement in such a manner as to regulate the amount of energy and duration of contraction required in each. The removal of this influence will not produce paralysis, or even paresis of movement, but will lead to irregularities in the energy and duration of contraction.

Now what will happen if the influence of the nucleus of Deiters is removed on one side? Under these conditions, if this view be correct, when the corresponding sixth nucleus is put in action, and both eyes directed to the same side, the contraction will be unsteady and there will be *nystagmus*. When the corresponding arm is moved there will be no paresis, but an unsteady and imperfectly regulated movement, such as the patient exhibited. In the same way, in walking, there will be an absence of perfect control of movement such as might lead to a stamping movement of the foot and occasional stagger to the corresponding side. In some cases the disorder of movement will be greater than that exhibited by my patient. In the leg, too, there will be no paresis.

The symptoms in the case described above corresponded so closely with the condition which in January I had indicated as likely to follow from interference with the function of the nucleus of Deiters, that there seemed no doubt that the tumour must be in a situation in which it could compress that nucleus. The fact that the first symptom was a left-sided nerve deafness suggested pressure on the auditory nerve. Now, the two situations in which tumours commonly occur which could press




To illustrate Dr Bruce's Paper.

FIG. 1.—SCHEME OF CONNECTIONS OF CEREBELLUM AND NUCLEUS OF DEITERS.

C.R. Restiform body.
SUP. VERMIS. Superior part of middle lobe of cerebellum.
S.F. Sagittal fibres connecting cortex of middle lobe with
R.N. The roof nucleus.
C.V.T. Cerebello-vestibular tract from roof nucleus to D. N.
D.N. Deiters' nucleus.
S.C. Semicircular canals.
VIII. Vestibular root of auditory nerve.
III. Third nucleus.
VI. Sixth nucleus.
P.L.F. Posterior longitudinal fasciculus.
V.S.T. Vestibulo-spinal tract from nucleus of Deiters to anterior cornu of spinal cord.

on the auditory nerve and on the side of the upper medulla, are:—the dura mater immediately behind and outside the internal auditory meatus, and the flocculus of the cerebellum. I have in my possession two specimens of tumours growing from the former site. They are fibro-cellular, ovoid masses, attached to the dura mater by a narrow peduncle. If they could be reached surgically, they appear to me capable of removal. The other site is the flocculus, in which I had never previously met with a tumour. Such a growth, however, is probably not removable. I know no criterion which would enable one to diagnose between these two sites, and inclined to the former as being more probable, owing to its greater frequency in my experience.

The patient while in hospital grew slowly more feeble, without for a time showing any special aggravation of his symptoms. In the beginning of May however, there was an attack of cerebral vomiting, a marked rise of temperature, a decided diminution of visual acuity, and an increase of hebetude—which indicated that a fatal termination was not far distant.

Mr Cotterill, who had previously seen the case with me, consented to operate in the hope of being able to reach the tumour. Very serious hæmorrhage from the emissary veins of the occipital bone, which was controlled with difficulty, greatly impeded the operation; and after the left hemisphere of the cerebellum was exposed, it bulged through the trephine wound to such an extent that the tumour could only be touched with the tip of the finger. It could not be removed without previous ablation of the left hemisphere of the cerebellum. After the operation, the patient, who appeared to be progressing fairly for three days, somewhat unexpectedly collapsed.

A *post-mortem* examination was granted, with the result that a vascular, fibro-sarcomatous tumour of the size of a walnut was found in the situation suspected, viz.—in the posterior fossa of the skull—adherent to the dura mater, immediately behind the internal auditory meatus. It had, however, its origin in the flocculus, and had involved the dura mater and petrous bone secondarily. The tumour had involved the glosso-pharyngeal nerve as well as the auditory, and had compressed a few of the fibres of the fifth nerve.

In this way the loss of taste, the deafness, and perhaps the anæsthesia of the left conjunctiva had been produced. The left facial nerve was also stretched over the tumour to such an extent that it is difficult to understand how the left facial paresis was so slight. The tumour had compressed the left anterior pyramid to a considerable degree, as is shown in Fig. 2, but it was evident, from the fact that the right arm and leg were unaffected, that the pyramidal fibres had been merely displaced and not destroyed. It was also evident that the inco-ordination of left arm and leg could not be ascribed to the pressure on the left pyramid, since that pressure acted on the medulla above the decussation of the pyramids, and therefore if it had any influence at all, should have affected the right side, and not the left, as was the case.

A cross section of the tumour and medulla showed that the position of the former was such as to exert pressure on the nucleus of the vestibular nerve (the nucleus of Deiters) and on the cerebello-vestibular tract from the middle lobe of the cerebellum to this nucleus.

In view of the now established connection of the nucleus of Deiters with the sixth nucleus and with the same side of the spinal cord, I would submit that we have now valuable data for the localisation of cerebellar disease, of the correctness of which the case submitted to you is a confirmation.

## Remarks on the Surgical Aspect of the Case

By J. M. Cotterill, M.B, F.R.C.S.Ed.

It is evident from a consideration of the symptoms in the above case, which Dr Bruce kindly asked me to see with him, that it was by no means a tempting one for surgical interference. It was only after full consideration of all the *pros* and *cons*, and with a complete realisation of the difficulties which would probably meet us that I undertook to operate. The points which decided me to act were—

1. The long history and chronic nature of the case, which indicated the probability of the tumour being of a benign character, such as fibrous or fibro-cellular.

2. The fact that the symptoms denoted that the tumour began in, and for a time was apparently restricted to, that part

of the base of the brain near the internal auditory meatus. One hoped that it might be a fibroma growing from the dura in this position, and possibly pedunculated.

3. The fact that during the last few days the symptoms had become suddenly and seriously worse, and that it seemed evident that death would shortly ensue, unless the tumour could be removed.

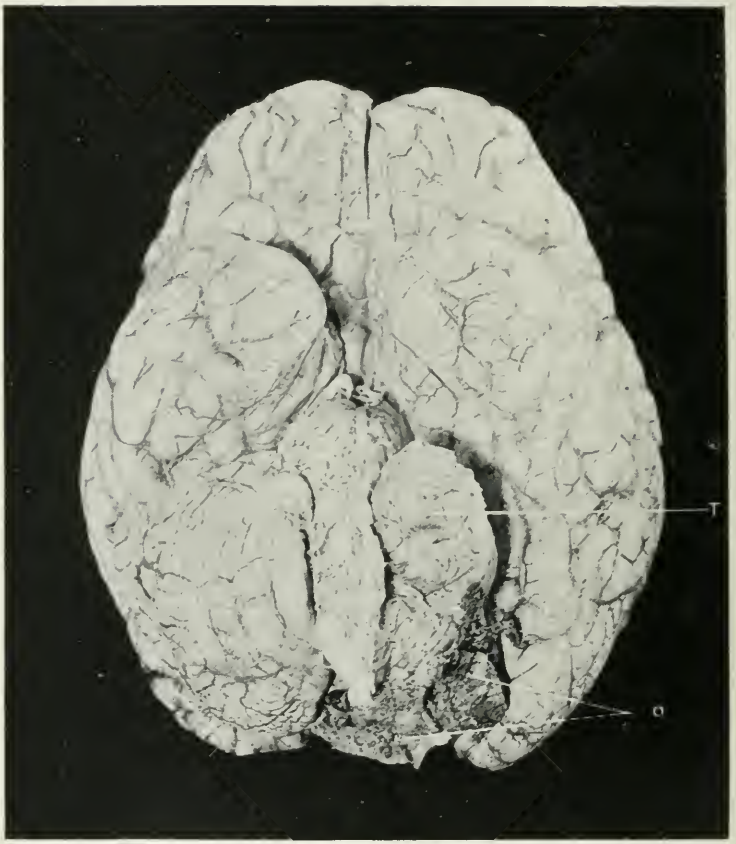
Having determined to operate, I satisfied myself on the cadaver that it was possible to remove such a tumour as we hoped this to be by removal of a considerable part of the occipital bone below the attachment of the tentorium cerebelli.

This method was found at the time of the operation to be a good one by which to reach a tumour situated at the internal auditory meatus, and I was able by pushing the cerebellum towards the middle line to pass my finger between the dura and the cerebellum along the posterior surface of the petrous bone almost as far as its internal extremity. Unfortunately, however, this tumour did not spring from the dura, nor was it pedunculated nor encapsulated, but was in the flocculus, and had involved the dura and petrous bone secondarily. In order to gain access to the posterior aspect of the petrous bone it is necessary to take away a large part of the occipital bone below the level of the lateral sinus on both sides of the middle line, removing at the same time about one-third of the bony ring forming the foramen magnum. This is necessary in order to give room for the cerebellum to be pushed away towards the middle line, and so to afford space for the necessary manipulations.

I made a curved incision from behind one mastoid process across the back of the skull to a similar point on the opposite side, and turned down a large skin and muscle flap. The trephine was then applied to the occipital bone on each side of the middle line, and the intervening bone removed by gouge forceps, great care being taken to protect the underlying central sinus by gently insinuating a hernia director between it and the bone.

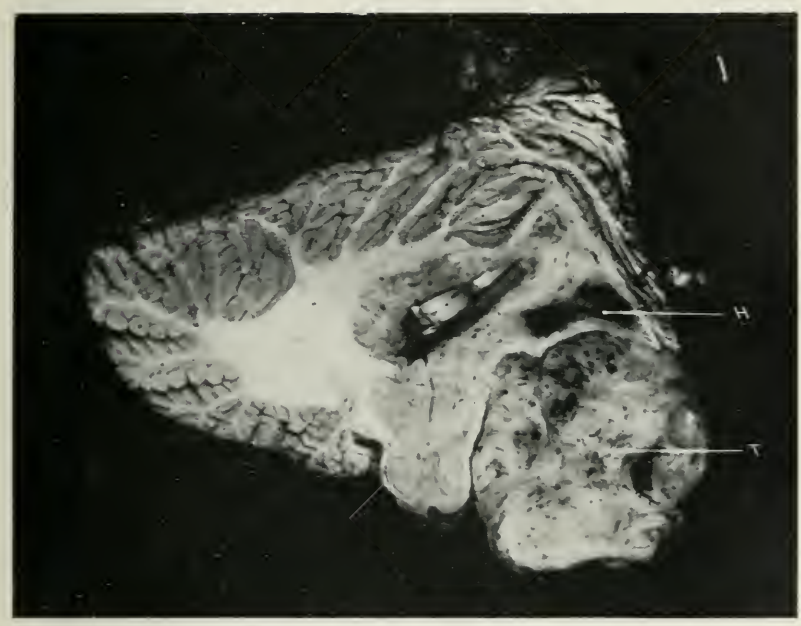
I have found in several such cases that in spite of the removal of such a large part of the occipital bone, the cerebellum is well supported afterwards by the thick muscle and skin flap which is sewn back in position.

The hæmorrhage in this particular case was very severe.




To illustrate Dr Bruce's Paper.

Fig. 2.—Photograph of base of brain showing the tumour, T, in the flocculus and the degree of pressure exerted by it on the medulla and pons.
O, the portion of the cerebellum injured in attempt to reach the tumour.




To illustrate Dr Bruce's Paper.

Fig. 3.—Transverse section of pons, cerebellum and tumour.
T, the tumour.
H, a recent hæmorrhage into the white matter of the cerebellum.
The amount of pressure on the side of the upper medulla is well shown.

It arose chiefly from the emissary veins which were unusually large, and, owing presumably to the pressure exerted by the tumour on the sinuses at the base of the brain, were extremely engorged with blood. Neither plugs of gauze nor Horsley's paste were of the slightest avail, and the use of firm wooden plugs was necessary to enable one to proceed with the operation. In another such case I should be inclined to operate with the head very considerably raised above the level of the trunk and limbs.

Having failed to make out the tumour distinctly by passing the finger between the dura and cerebellum, I gently insinuated the little finger into the substance of the left lobe and found an area of distinctly increased resistance at its anterior aspect. The tumour being evidently in the substance of the cerebellum, no further attempt was made to remove it. Had it been fibrous and encapsulated, I believe it might have been removed satisfactorily.

The patient went on very fairly well for three days, and then died rather suddenly and unexpectedly, after having four convulsive seizures, chiefly affecting the right side. He was conscious till ten minutes before death.

In order to avoid shock I should have preferred to have operated in two stages, but the urgency of the symptoms made this impossible.

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## 2. SOME OBSERVATIONS ON THE DEVELOPMENT OF THE TESTICLE.

By James Foulis, M.D., F.R.C.P. Ed.

In order to trace out in a successful and satisfactory manner the various steps in the development of the testicle, it is necessary to be acquainted with the main facts in the development of the ovary, as the study of the development and growth of these two organs becomes more interesting and important, just in proportion as the homologies between them are laid bare in the various stages of their development.

Thus at once arises the question, is the ovary a tubular structure in the same sense that the testicle is? Then, too, as regards the theory of spermatogenesis, are the spermatozoa

derived from the nuclei of cells, just as are the germinal vesicles of the ova? and are the cells which give origin to the spermatozoa in the male, and to the germinal vesicles in the ova, both derived from the germ epithelial cells in the early embryonic condition? These and many other interesting questions at once arise as we study side by side the development of the testicle, and the development of the ova and the ovary.

Since the observations of Pflüger and Waldeyer on the development of the ova and the ovary were published, all observers are agreed that the first trace of the ova is among the germ epithelial cells, and that the germinal vesicles of the ova are derived from the nuclei of the germ epithelial cells; but all are not agreed as to the manner in which these germ cells become enclosed in the meshes of the ovarian stroma. Pflüger described the germ epithelial cells as growing downwards into the mesoblastic stroma in the form of columns of epithelial cells which enclose among them some of the primordial ova. These columns are the "egg tubes" of Pflüger. These "egg tubes" become broken up into rounded groups or "cell nests" by the continued in-growth of the stroma, and one or more cells in each group becomes a primordial ovum, the other cells remaining small to form the epithelial lining of the young Graafian follicles thus formed. According to this view, the ova and the cells lining the Graafian follicles are both derived from germ epithelial cells on the surface of the young ovary which grow downwards in the form of tubular columns into the subjacent vascular stroma. This view makes out that the young ovary is a tubular structure at an early stage of its development, just as the testicle is, and that the epithelial lining of the primitive tubules, and of the subsequent Graafian follicles, is from germ epithelial cells which do not develop into ova. Other observers hold that the germ epithelial cells do not grow downwards into the ovarian stroma in the form of tubes, "the egg tubes" of Pflüger, but that from the very first trace of the ovary, the vascular stroma grows outwards, and sends its young processes among the germ cells enclosing them in its meshes in groups of various sizes, and that the Graafian follicles are formed by the stroma enclosing one or more primordial ova, along with a number of smaller germ cells, which become the epithelial cells lining the young Graafian follicles.

Another view is that published by myself more than twenty-five years ago in my graduation thesis "On the Development of the Ova and the structure of the Ovary in Man and other Mammalia." My conclusions were that the ova are derived from the germ epithelial cells, and that the nuclei of such cells become the large germinal vesicles of the ova, and that from the very first trace of a thickened germ epithelial layer on the surface of the young ovary, the young vascular stroma sends its processes outwards among these cells enclosing variously-sized and variously-shaped groups of them in its meshes, and that it is by the continued onward and outward growth of this stroma among the individual cells in each group that the Graafian follicles are formed. According to my view, all the included germ cells may become ova. By the growth of the vascular stroma not only round the groups of cell nests, but between and around the individual cells in the nests, the whole ovary at last becomes a mass of young ova enclosed in the meshes of the stroma, and at last each germ cell is enclosed in a single mesh of stroma, this last mesh becoming the young Graafian follicle. But, further, in accordance with this view, the cells lining the young Graafian follicles are derived from the cells of the vascular stroma, for I discovered that, even among the germ epithelial cells on the surface of the ovary, whenever a primordial ovum is seen it is found to be enclosed in a mesh of ovarian stroma. In all parts of such a young ovary each primordial ovum is found to have round its protoplasm a mesh of stroma; and so invariably is this the case, that I was forced to the conclusion that this capsule of young vascular stroma is the direct exciting cause of growth and development of each primordial ovum, and that nutriment is conveyed to such growing ova by the cells in the vascular stroma itself. Here, then, we have the conclusion that while all the ova are derived from germ epithelial cells, and that the Graafian follicles are formed by the ultimate meshes of the vascular stroma around the ova, the cells lining the Graafian follicles are derived from the stroma, and not from the germ epithelial cells, as was described by Pflüger, Waldeyer, and other observers; and as regards the theory of the tubular structure of the ovary, I came to the conclusion that the so-called tubes are only microscopic appearances seen in sections of variously-sized and variously-shaped groups of included cell

nests. Since these conclusions were published, although I have followed pretty closely the recent literature on the subject, I have seen and read nothing to cause me to alter my views in any way.

Whether the "egg-tubes" of Pflüger do or do not take part in the formation of the Graafian follicles, it is certain that in the ovaries of a child at birth, there are not less than 70,000 included ova: but how few of these come to maturity!

It is interesting to observe that in all mammals the ovum can only escape from a ripe Graafian follicle by bursting through the wall of the follicle, and that it would at once fall into the peritoneal cavity were it not for the fimbriated extremity of the Fallopian tube which catches it and conveys it into the cavity of the uterus.

While not allowing that the ovary is a tubular structure in the sense in which Pflüger described it, there can be no doubt that the testicle is from the first stages of its existence a tubular structure, and that its seminal secretion with its living spermatozoa is produced within its tubes, and that this secretion can only escape from the organ by the vas deferens, a very long tube, derived from the Wolffian duct, which in the embryo is connected at its lower end with the cloaca.

While it is thus clear that the ova are derived from the germ epithelial cells, and that the Graafian follicles are the ultimate meshes in the ovarian stroma and lined with cells derived either from the germ epithelial layer or from the cells in the stroma, it is by no means clear how the tubes of the testicle are formed, and from whence are derived the cells lining those tubules from which are produced the living spermatozoa.

The subjects of ovulation and spermatogenesis can only be cleared up by knowledge of the minute structure both of the ovary and testicle, and this can only be acquired by tracing out the development of these organs from their earliest appearances in the embryo; and even then we have not begun at the beginning of the study, for it is absolutely necessary to trace back to their origin from the three primitive germinal layers all those structures which play so important a part in the later development of the organs. Is the germ epithelium, for instance, derived from the epiblast or from the mesoblast or from the hypoblast? Are the Müllerian ducts

and the Wolffian ducts lined by cells derived from the epiblastic germinal layer? If the epithelium lining the Graafian follicles and the tubules of the testicle can only be produced from epithelial cells, it is most interesting to find out if the parent cells are of epiblastic origin. Much has been done by Kölliker and later observers to show that the Wolffian duct is derived from the epiblastic germinal layer, and according to Balfour and others the Müllerian ducts also have an epiblastic origin. These are points, however, which I only touch upon in passing as being of the greatest interest. My own investigations start from the already formed germ epithelium and from the already formed Müllerian and Wolffian ducts, all of which it is possible to see in the fresh and microscopic preparations in my possession.

The question of the origin of the tubules of the testicle and as to the origin of the cells lining those tubules is of the greatest interest. In the last edition of Quain's "Anatomy," vol. i. part i., Embryology, at page 126, there is a drawing representing a section of the germinal epithelium and adjacent stroma in a male chick embryo, taken from Semon, in which it is attempted to show that strands of cells growing up from the Wolffian body towards the germ epithelial layer, come in contact with enlarged primordial ova and cell nests, and that from these cell nests and primordial ova the epithelial cells lining the tubules of the testicle are formed, although all stages of the process have not been observed. If this origin of the cells lining the tubuli seminiferi should turn out to be correct, then, of course, there is established a most interesting homology between the germinal vesicles of the ova and the nuclei of the cells lining the tubuli seminiferi; for it is from these nuclei that the heads of the spermatozoa are supposed to be produced.

There certainly is a stage of development in the testicle, as is the case with the young ovary, when the whole gland seems to consist of a thick layer of epithelial cells capping a little outgrowth of stroma from the Wolffian body. It is at this stage that is impossible to say if the gland is male or female. In the case of the testicle, if there is at any time a connection between the germ epithelial cells and tubular outgrowths from the Wolffian body, it must be before the strong fibrous capsule or tunica albuginea of the testicle is formed, for this structure entirely shuts off the tubules from the outside world. Anterior

to the formation of this capsule there may be some connection between germ epithelial cells and tubules from the Wolffian body, but I know of no drawing which proves this, and I have not seen anything of the kind, though both in the young ovary and testicle it is possible to see certain tubular structures entering these glands at their base. In the testicle, as I shall presently show, this growth of tubules goes on until the whole capsule of the testicle is distended with tubules, which, according to my investigations, grow outwards from the Wolffian body, and do not at any time grow downwards from the germ epithelial layer. In the case of the young ovary, though tubules from the Wolffian body enter at the hilum and grow to a certain distance, they gradually abort, and take no part in the structure of the ovary itself.

My present researches were undertaken with the object of tracing out the origin of the tubuli seminiferi, and, if possible, of the cells lining them, from which are produced the spermatozoa. There is something very fascinating in the study of the development of the ova and of the spermatozoa; but this latter cannot be successfully done without first of all discovering the origin and source of the tubuli seminiferi.

Knowing that the vas deferens in the adult is represented in the embryo and fœtus by the Wolffian duct, it was necessary, in the first place, to find out the exact position of the Wolffian duct in its relation to the testicle in its earliest stages of development; but this was a difficult matter, because in glands so young it is not possible to say if we are dealing with the male or female organ.

Two or three years ago I discovered that the embryos of deer show the Wolffian bodies in a remarkably clear manner, and so I secured a number of them at different ages, and having prepared them, it was possible to study the Wolffian bodies both in fresh preparations and subsequently in microscopic sections. In an embryo of a deer, less than an inch in length when fully pinned out, it is possible to clear away the liver, and thus expose the Wolffian bodies. In such a young embryo the yolk-sac is still attached to the primitive intestinal tube, and the anterior and posterior limbs stand out as spade-like buds. The two Wolffian bodies may thus be seen like two long thick sausages, lying side by side on either side of the central mesentery and intestinal tube, and extending from the

site of the anterior limbs backwards to the pelvic region of the young animal, where, even at this early age, the two Müllerian ducts can be seen joining each other in the middle line (Plate vi. Fig. 12). The Wolffian bodies thus appear as two large reddish-brown sausage-shaped bodies, tapering off slightly at their ends. Under the microscope a white streak-like ridge, E, can be seen on the inner side of each Woffian body, almost in contact with the central mesentery, and extending up and down the Wolffian body for fully three-quarters of the length of each body. This white streak is the primitive germinal ridge, and in connection with this at its upper part both the ovary and testicle make their first appearance. I would particularly call attention to the upper end of this germinal ridge. In its connection with the Wolffian body it does not end abruptly, but seems to enlarge slightly, and then fade off into the substance of the Wolffian body.

It is important to notice this part, for it is here that we have the first appearance of the young testicle as a distinct body. There is here always an important connection between the young testicle and the Wolffian body. At its lower end the germinal ridge gradually fades off, and is in direct connection with the peritoneum which covers the lower end of the Wolffian body and other organs in the pelvic region. The germinal ridge may be looked upon as a fold of thickened peritoneum projecting out from the peritoneum covering the Wolffian body on its inner aspect, and at the lower end of the germinal ridge this peritoneum is in continuation with the general peritoneal layer which covers all the pelvic organs and parts in that region.

Turning now to Plate vi. Fig. 12, the two Wolffian bodies D D are well seen, and the germinal ridges E E are seen as described; and in Fig. 13, which is a more highly magnified drawing of one of the Wolffian bodies, the germinal ridge E may be seen enlarged at its upper end, and gradually fading off into the substance of the Wolffian body at its upper part B. The letter C points to the Müllerian duct.

In transverse sections the Wolffian bodies present the appearance as seen in Fig. 14, which is from a microphotograph of a section through the body of such an embryo as is depicted in Fig. 12.

In such a section the neural canal A is seen, and at F the

chorda dorsalis is clearly observed, while the letter M points to the aorta, full of blood corpuscles. The two Wolffian bodies D D are seen on each side of the aorta ; and it is interesting to note that the glomeruli occupy a situation in front and to the side of the aorta, from which they receive their blood supply, while the tubular parts of the Wolffian bodies lie to the outside of the aorta behind the situation of the glomeruli. The letter C points to the Müllerian duct in each Wolffian body. I would here like to call attention to the fact that in several of my preparations the lumen of the Müllerian duct can be seen in direct connection with the lumen of some of the tubes in the Wolffian body. In other words, my preparations show that in such an embryo the Müllerian duct is not a simple tube, but receives tubules from the Wolffian body. This observation may be of some importance in connection with the origin of the epithelium lining the Müllerian duct from the epiblast.

In Fig. 14 the young germ glands, ovaries or testicles are indicated by the letters B B on each side of the central mesentery G ; while the glomeruli receiving branches of blood supply from the aorta M are indicated by the letters E E.

So much then for the early appearance of the Wolffian bodies as seen in such a young embryo. These drawings have been most faithfully made, and correctly represent the appearances seen in such a young deer embryo.

Bearing these appearances in mind, let us look more closely at the young germ glands. They appear to the naked eye as two almost semicircular outgrowths from the Wolffian bodies on their inner side, B B, and project towards the mesentery in the middle line.

Under high powers of the microscope the young gland is seen to be a growth of stroma from the Wolffian body, capped over with a thick layer of epithelial cells, which are directly continuous with the peritoneal cells on its margins. Numerous blood-vessels, filled with nucleated embryonic blood corpuscles, can be seen in it, going up as far as the layer of epithelial cells on its surface ; but it is at this stage impossible to say if such a gland is an ovary or testicle.

But there is a stage in the development of the genital glands at which it is possible to distinguish between the ovary and testicle. As soon as the young gland becomes distinctly globular the difficulty is at an end. For by means

of a sharp razor it is now easy to slice off a minute piece of the gland in question ; and if such a piece is torn to shreds in a little water by means of needles, and then examined under the microscope, if it is an ovary the sliced-off piece will be found to consist of cells of various sizes, permeated in all directions by very vascular stroma, but without the slightest appearance of an enclosing fibrous capsule. If the gland in question is a testicle, there will always be found some trace of an enclosing peritoneal capsule. The testicle at this stage is always firmly attached to the Wolffian body at its upper part ; and if such a gland is sliced in half, that half which is still attached to the Wolffian body, may be placed under water and cleared out of its contents by means of fine needles ; and there remains a beautiful fibrous capsule. It is impossible to treat the half of an ovary in this way, as the organ, being very brittle and cellular, cannot be so dissected, and it has no fibrous peritoneal capsule, as is the case with the testicle.

It was while treating young ovaries and testicles in this way in my efforts to determine which was which that I made the following observation.

The delicate little capsule, which in the case of the testicle always remained after its contents were cleaned out, was found to be firmly attached by a stalk-like process of peritoneum to the peritoneal covering of the Wolffian body. Invariably I found a minute hole in the capsule thus left, and this hole had a distinct relation to the stalk of peritoneum attaching the testicle to the Wolffian body. Into this hole I could pass a small bristle, which, if pushed on, would go through the centre of the stalk connecting the capsule of the testicle to the Wolffian body.

If you now look at Plate ii. Fig. 4 the letters B B point to minute holes in the capsules of the two testicles which have been sliced in half and cleaned of their contents. This Fig. 4 represents the appearance seen on looking into the body of a fœtal deer, not more than two inches in length, after it had been prepared in such a way as to show the two Wolffian bodies D D. In the sliced capsules of the young testicles, the letters B B point to the minute holes in the capsules, as seen from the inside view. This hole is always seen in exactly this same situation in such young capsules, and as it is always present it must be of some

importance. A full description of this figure is given in the proper place; but what I have indicated suffices to show that the young testicle at this early age always possesses a firm fibrous capsule, which if cleaned out will always show in the same position as indicated in the drawing a minute hole through which some structures must pass from the Wolffian body into the capsule of the testicle. No other hole can be discovered in the whole capsule of the testicle; and it would appear that the entire tubular contents of the testicle come into the capsule of the organ through that minute hole.

In order that this minute hole and its relation to other parts may be more fully studied, we have prepared Plate iii., which is a large drawing representing the Wolffian body D with the young kidney K, and the sliced capsule of the young testicle H, in which the letter B points to the hole in the capsule as seen from its inside.

The Müllerian duct C is well seen, and folds of peritoneum A pass up from the end of the Müllerian duct and Wolffian body towards the diaphragm of the young animal. A large part of the capsule of the testicle has been sliced away in order to show the hollow in which the testicle lies between the lower part of the kidney K and the upper part of the Wolffian body D.

Stretching down from the lower part of the testicle H is a strong band of a fibrous nature G, the gubernaculum testis, which passes down in continuity with the peritoneal layer which spreads over the lower end of the Wolffian body and the string F formed by the junction of the Müllerian and Wolffian ducts at their lower ends. This drawing has been most faithfully drawn by means of a binocular microscope, and is true to nature; but to understand it the more clearly, we must now look at Fig. 3 Plate ii. which represents the appearances seen in a fœtal deer considerably younger than figure 4 represents.

In Fig. 3 we have faithfully represented the two kidneys K K, the two testicles B B, and the two Wolffian bodies D D, as seen by means of a binocular microscope on looking into the body of a fœtal deer, less than two inches in length, prepared by a most careful dissection to show all such parts in their natural positions. I shall not refer to the other structures repre-

sented in the meantime, but the proper description of this important drawing will be found attached to the plate. What I wish particularly to direct attention to is the stalked condition of the young testicles B B, at this stage of their development. The letters X X point to the stalks by which the testicles are attached to the Wolffian bodies at their upper part. These stalks of connection are seen in all young testicles at this age; and on looking at Fig. 4 again, which, as already mentioned, is drawn from the appearances seen in the body of a fœtal deer somewhat older, it will be noticed that the stalks are not visible, the reason being that here the testicles are much larger and more globular, and thus hide from view the stalks of connection through the centre of which pass certain structures. In the case of the younger animal as seen in Fig. 3, the testicles are more elongated and less globular, and thus their stalks of connection, X X, with the upper end of the Wolffian bodies are not only well marked but are well seen. I would here like to remark that this entire drawing has been made with the greatest care and attention to all points, and it very faithfully represents the parts described.

Having thus, by means of these figures, directed attention to the stalk of the peritoneum which connects the young testicle with the upper part of the Wolffian body, and to the minute holes seen within the capsules of the testicle always at the same spot, which undoubtedly give passage to some structures passing from the Wolffian bodies into the capsules of the testicles, let us now in the next place try to find the exact position of the Wolffian duct and its relation to the testicle and Wolffian body.

Where is the Wolffian duct to be found? A part of the Wolffian duct can always be found at the lower end of the Wolffian body, where in conjunction with the pelvic end of the Müllerian duct, it passes down to become attached to the cloaca. The two Müllerian ducts as they course downwards along the outer borders of the Wolffian bodies, at last meet each other in the middle line in the pelvic region. In the female this junction of the lower ends of the Müllerian ducts results in the formation of the body of the uterus, and the Müllerian ducts in the upper parts become the Fallopian tubes. The two Wolffian ducts join the two Müllerian ducts as they pass down behind the allantoic stalk and the four ducts

together form the genital cord, or string, which passes down to be connected with the sinus urogenitalis at the base of the allantoic stalk. But it is at its upper part that the Wolffian duct is so difficult to find. That part of the Wolffian duct which becomes the epididymis and the vas deferens is very difficult to discover. After long seaching I discovered the upper part of the Wolffian duct in the following way. See Plate i. Fig. 1. Here is represented the Wolffian body D, with the kidney K and the testicle B and the Müllerian duct C, and the gubernaculum testis G. On the right side the Wolffian body has been cut in half and its exposed cut surface M is seen. At the lower part of the same figure the letter E points to a string formed by the Müllerian duct in front and the Wolffian duct behind. These two ducts join the same ducts coming down from the right Wolffian body at H, and form the genital cord or string. The letter N here points to a torn surface, a point at which the allantoic stalk was attached to the genital cord, and at L can be seen a structure which is evidently the verum montanum prominence, and supposed to be the homologue of the hymen in the female. Now if the right Wolffian body is thus cut in half and the genital cord thus torn away from the allantoic stalk, the left Wolffian body with the testicle attached to it may be then lifted up and turned right over on to its side.

If this is properly done, then we bring into view the appearances seen in Fig. 2, which represents the left Wolffian body lying on its side in such a way as to expose the torn mesorchium E E and the under surface of the left testicle B, and the gubernaculum testis G is well brought into view; under low powers of the microscope a tubular structure H H can now be seen passing upwards from under cover of the upper border of the mesorchium towards the testicle with which it comes into intimate relation at the spot indicated by the letter H. The letter L points to a small cyst which springs from the Wolffian duct before it reaches the testicle. The Wolffian duct thus exposed lies under the peritoneum covering the Wolffian body, and its own fibrous covering is in direct continuity with the peritoneal and fibrous capsule of the testicle. We must be careful not to mistake the gubernaculum testis for the Wolffian duct—between which there is no connection; but on looking down into the body of a young fœtal deer, this gubernaculum

testis as it leaves the lower end of the testicle covers both the Wolffian duct and Müllerian ducts at their pelvic ends, and may possibly be mistaken for the Wolffian duct. The Wolffian duct in its upper part cannot be seen except by turning over on to its side the whole Wolffian body with the testicle attached. There it is at once seen as in Fig. 2 as a rod-like structure H H quite apart from the gubernaculum testis G.

In Fig 1, the gubernaculum testis G is well seen, but the Wolffian duct cannot be seen in this front view of the Wolffian body and testis.

How does the Wolffian duct become attached to the testicle at its upper part?

The Wolffian duct is at its upper part firmly connected with the capsule of the testicle—see Plate iv. Fig. 6—which represents the capsule of the testicle firmly attached by the peritoneal stalk to the upper part of the Wolffian body. The letter B points to the hole in the capsule which passes through the centre of the stalk. Fig. 7 represents the Wolffian duct C attached to the capsule of the testicle B. The Wolffian body is lying on its side with its under surface exposed to show the Wolffian duct C. The fibrous capsule of the testicle is continuous with the fibrous covering of the Wolffian duct. Fig. 8 shows the capsule of the testicle B partly torn away from the stalk of peritoneum H. The Wolffian duct still atached to the capsule of the testicle is also partly torn away from the Wolffian body as seen at the letter M. In Fig. 9, we see in the clearest manner that when the capsule B of the testicle is partly torn away from the stalk H, the Wolffian duct goes with it, and that the Wolffian duct is actually torn away from the Wolffian body as is seen in the torn surface M. And just at the spot on the capsule where it joins the stalk from the Wolffian body there can be seen a scar L indicating a torn surface; the capsule of the testicle has been torn through at this point. The study of the Figs. 8 and 9 leads us to the conclusion that the capsule of the testicle and the covering of Wolffian duct are continuous with each other, just as the bowl of an ordinary clay pipe is continuous with its stem.

Much has been said about the stalk which connects the capsule of the testicle with the Wolffian body at its upper part. It is only at a certain stage of development that this stalk can be so well seen, as is shown at X in Fig. 3 Plate ii. Although

it is always present at a certain stage, and can always be seen before the testicle has enlarged and become globular, it is gradually hidden from view behind the enlarging testicle, and so may be overlooked ; but it is always present as an essential part in the development of the testicle.

We must now go to the microscope to tell us the nature of this stalk, and the nature of those structures which pass through its centre into the capsule of the testicle. It is possible to make serial sections through the testicle and Wolffian body in various directions as they are naturally and firmly attached to each other by that stalk ; and fortunately I have succeeded in making such sections which pass vertically downwards through both testicle and Wolffian body and through the stalk which connects them, and also transverse sections, in series, through the Wolffian body and testis at the same part.

The first thing that is noticed in viewing such a vertical section is the strong fibrous capsule of the young testicle (see Figs. 10 and 11, Plate v.). The letter A points to this strong capsule in each figure. Nothing of the kind exists in the sections of an ovary at this same stage of development ; and on looking carefully at Fig. 10 it will be seen that the whole tubular structure of the testis B is enclosed within the fibrous capsule, and that it has no outlet from the organ except by a minute aperture which passes through the centre of the stalk X, so often referred to. In Plate ii. Fig. 4, these minute apertures in the capsules of the testicles are well seen at the spots indicated by the letters B B. In Fig. 10 Plate v., which is now before us, the microscope has shown to us that in nature this minute aperture is occupied by a complex cellular column (see X, Fig. 10), which passes through the stalk centre. This solid cellular column X can be seen coming up from the Wolffian body and passing through the centre of the peritoneal stalk right into the capsule of the testicle, and as soon as it is well within the capsule of the testicle it gives out branches of cells in all directions as it grows forward in the long central axis of the testicle. It grows so far forward that it almost comes in contact with the enclosing capsule at the extreme end of the gland ; cellular branches spring out in all directions from it, until the testicle capsule is tightly distended with these rudimentary tubuli seminiferi. I have described the central column of cells which passes from the Wolffian body into the

testicle as a solid cellular column. Under very high powers of the microscope it is possible to see in the substance of this cellular column appearances which can only be described as transverse sections of minute tubules. The branches of cells which radiate in all directions from this central cellular column butt up closely against the capsule of the testicle, and on careful examination under high powers of the microscope numerous transverse sections of minute tubes can be seen in all directions, and it is quite evident that all these cellular offshoots from the central column have become minute tubules and are all in direct communication with the solid column of cells from which they have outgrown. Each small tube consists of a basement membrane on which is a single layer of more or less columnar nucleated cells. All these tubes are blind at their ends as far as can be seen, and the epithelial cells lining these numerous tubes are without doubt derived from the central cellular column. The central cellular column as it passes into the testicle is surrounded on all sides by a thick coating of peritoneum which constitutes the stalk, and this stalk can be seen to give rise to the whole capsule of the testicle, which is now distended with the tubules which have sprung out in all directions from the cellular column X. On tracing the central cellular column back from the testicle towards the Wolffian body we find it goes back through the centre of the stalk until it ends in the immediate neighbourhood of a number of cut tubules, as indicated by the letters H H H in Fig. 10, and this neighbourhood is the very spot towards which we have traced the end of the Wolffian duct; and there can be no doubt that the cut tubules as represented by the letters H H H in Fig. 10, and by the same letters in the transverse section, Fig. 11, are the cut tubules of the Wolffian duct with which the cellular column is in direct continuity. All these parts are, of course, seen in transverse section; but an examination of a whole series of such preparations, both vertical and transverse, shows that the convoluted terminal end of the Wolffian duct is in direct continuity with that central cellular column X, which enters the testicle through the centre of the peritoneal stalk, and which, as soon as it is within the capsule, gives out branches of tubules in all directions until the capsule itself becomes globular from such distention.

The Wolffian duct in its whole course from the testicle to

its pelvic end is in connection with the tubules of the Wolffian body. It receives branches of tubes from the Wolffian body along its course, especially at its lower end; and at its upper end it receives, either directly or indirectly, the whole tubular structure of the testicle by means of the cellular column which passes through the peritoneal stalk from the capsule. In all probability this central cellular column X gives rise later on to the vasa efferentia, which are in direct continuity with the convoluted tubules of the epididymis.

In Fig. 11, which is a transverse section of the young testicle, the central tubular column X is well seen giving out its tubules in all directions, and the letters H H H point to cut tubes with which the column X is undoubtedly continuous. As these figures, 10 and 11, are carefully drawn from microphotographs, their accuracy may be relied on.

Let us now, in conclusion, follow the steps in the development of the testicle from the first appearance of the gland at the upper and inner part of the Wolffian body.

The Wolffian body in the early embryo has already been described, and in Plate vii. Fig. 15 this is again represented in order that the primitive germinal ridge G may be seen at its upper part B swelling out, and fading off into the substance of the Wolffian body. It is at this point that the young testicle makes its first appearance as an outgrowth from the Wolffian body. In Plate vi. Fig. 14 shows in transverse section through the body of a young embryo the Wolffian bodies and the first trace of the genital gland, to which the letters B B point. On comparing this young gland with Fig. 13 or with Fig. 15, it is quite clear that the young gland is an outgrowth of vascural stroma from the Wolffian body, capped by a thick layer of epithelial cells. The whole primitive germinal ridge may be looked upon as a thick fold of peritoneum projecting from the inner aspect of the Wolffian body; but at its upper end certain structures grow into its substance from the Wolffian body, and in that situation the epithelial cells covering this germinal ridge become thickened as a layer over that part. The next step in the development of the testicle is the distinct bulging forward of the young gland as an elongated body having a stalk-like connection with the Wolffian body. In Fig. 16, Plate vii., we have a faithful representation of the young testicle as it thus appears. Microscopic sections of such a young

testicle show that its stalk X consists of thickened peritoneum, and that through the centre of the stalk passes a solid-like column of cells, from which spring out in all directions minute tubules, the future tubuli seminiferi of the adult testicle, and all these tubules are enclosed within a fibrous capsule of peritoneum continuous with the thick stalk itself. Here, then, we have clear evidence that the young testicle is a sub-peritoneal outgrowth from the primitive germinal ridge, pushed out, as it were, into the form of an elongated body by certain continually growing tubules which pass into its base from the Wolffian body through the centre of a stalk-like connection of peritoneum. I speak of it as a sub-peritoneal outgrowth, because sections show that the essential tubular structure is sub-peritoneal from the first. The capsule of the testicle is formed out of the primitive germinal ridge, which is itself at first a thickened fold of peritoneum on the inner aspect of the Wolffian body, as already described. It is now very interesting to follow the next step. As the testicle continues to grow and enlarge, it drags forward with it that part of the primitive germinal ridge which is immediately below it, and with which it is continuous; for it must be borne in mind that the capsule of the testicle and the rest of the germinal ridge are peritoneal structures, and both parts of the primitive germinal streak, so well seen in the early embryo. That part of the germinal ridge immediately below the capsule of the growing testicle becomes the gubernaculum testis G, and, as a fold of peritoneum, it passes down and fades off into the general peritoneal layer which covers the pelvic organs and lines the general cavity of the pelvis later on.

As the testicle thus becomes larger and more globular, its stalk-like connection with the Wolffian body becomes hidden from view. The stalk becomes much shorter and almost disappears as the capsule is distended outwards in a centrifugal manner by the continued growth of the tubuli seminiferi within it. In the figure, 17, the stalk cannot be seen though a fold of peritoneum is seen passing upwards off the surface of the testicle to become continuous with the peritoneum covering the body cavity of the embryo.

To establish the position of the Wolffian duct and its relation with the testicle at the very early stages of development is most important, bearing in mind that it is this duct, as the

vas deferens, which carries off the secretion of the tubuli seminiferi.

Looking down on the upper surface of the testicle and Wolffian body, as in Fig. 17, the Wolffian duct cannot be seen, but on raising up the Wolffian body and turning it over on to its side, as in Fig. 18, the Wolffian duct H H is clearly brought into view, and it always holds this position as regards the Wolffian body and the testicle; and, lastly, it is quite evident on looking at Fig. 10, Plate v., that the tubuli seminiferi are all in direct connection with, and derived from the cellular column X, which passes through the stalk to be connected with the Wolffian duct.

That the central solid column X, which passes through the stalk into the capsule of the testicle, gives rise to the tubuli seminiferi cannot be doubted. It is for further researches to settle the question as to whether both the Müllerian and Wolffian ducts are of epiblastic origin. In several of my microscopic sections there is distinct evidence of a communication between the Müllerian ducts and the tubules of the Wolffian body which join the Wolffian duct itself.

In conclusion, while it is quite clear that the germinal vesicles of the ova are derived from germ epithelial cells, it has not yet been proved that either the cells lining the Graafian follicles or the cells lining the tubuli seminiferi have their origin from germ epithelial cells. But if it can be shown that the Wolffian ducts have an epiblastic origin, then both the tubules of the testicle and the cells lining them can be satisfactorily accounted for. If the tubules of the testicle, as I have attempted to show, are derived from the terminal end of the Wolffian duct, then it is most probable that the epithelial cells lining those tubules have the same origin as the tubules themselves. In other words, according to these observations, if the Wolffian ducts and the tubuli seminiferi are of epiblastic origin, then the cells lining the tubuli seminiferi are also of epiblastic origin.

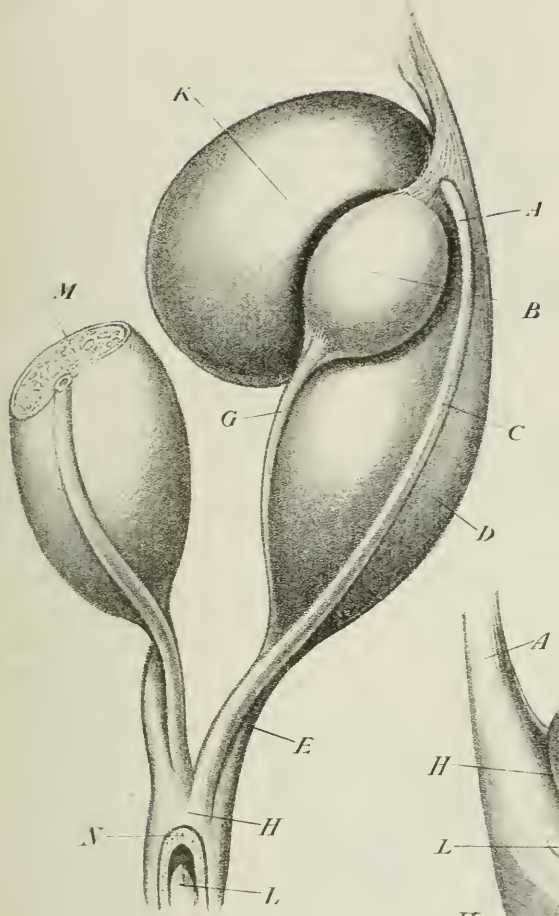



Fig. 1

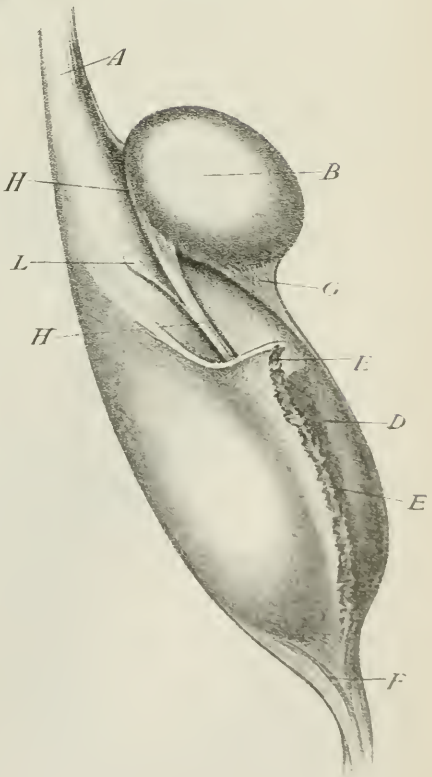
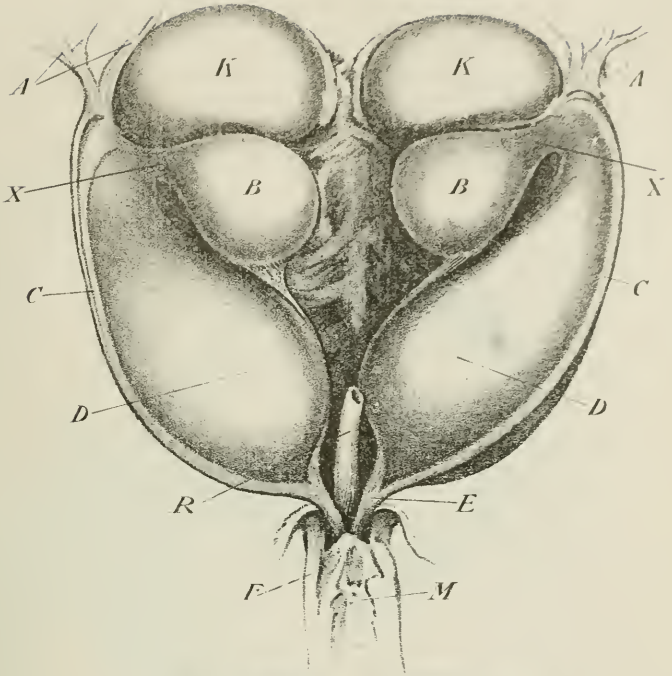



Fig. 2

Fig. 3



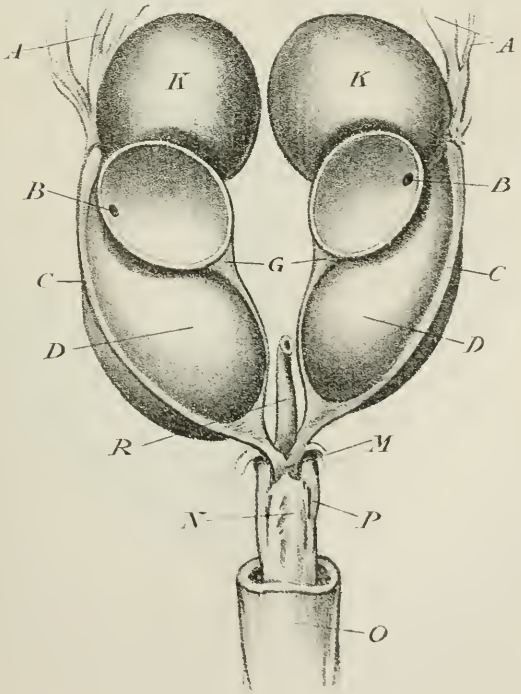




Fig. 4

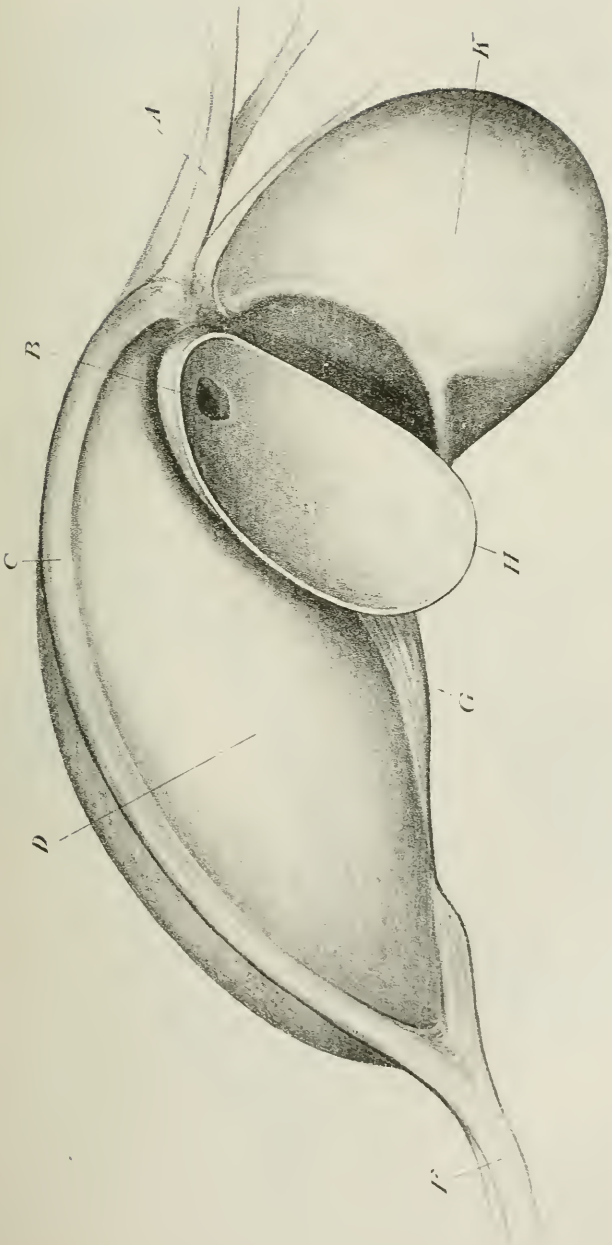



Fig. 5

Fig. 6

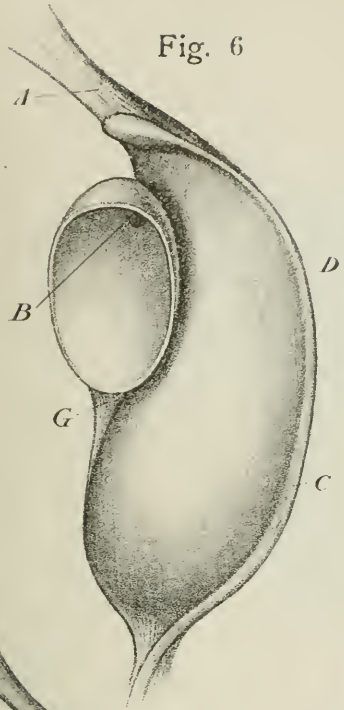
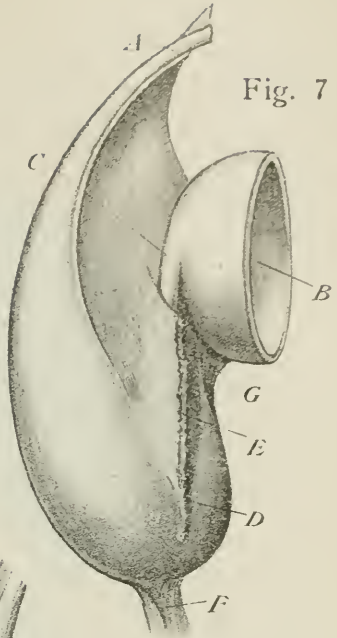
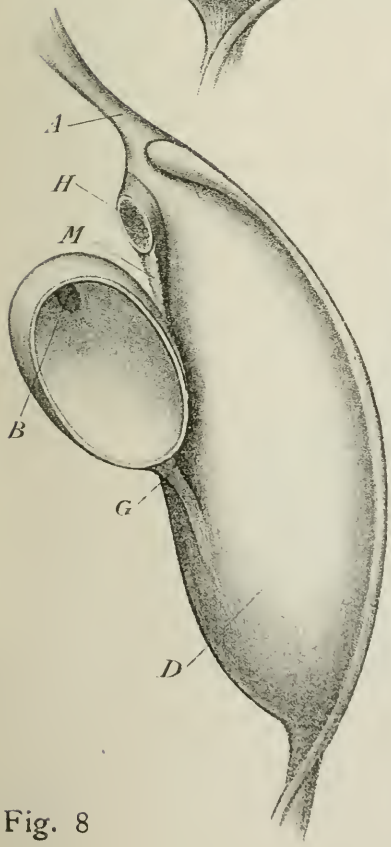



Fig. 7

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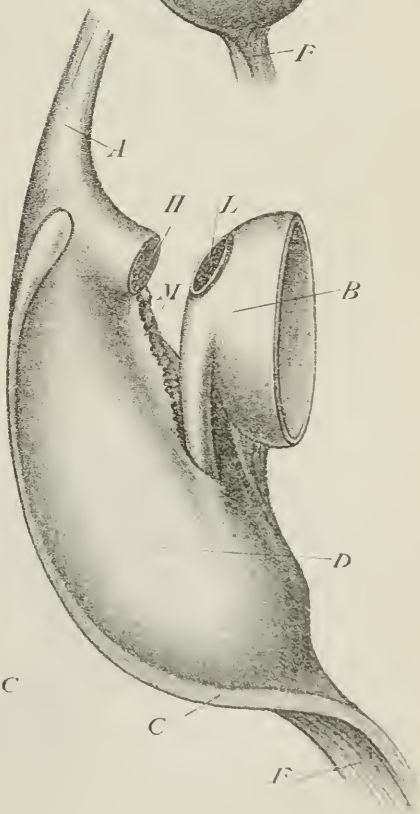



Fig. 9

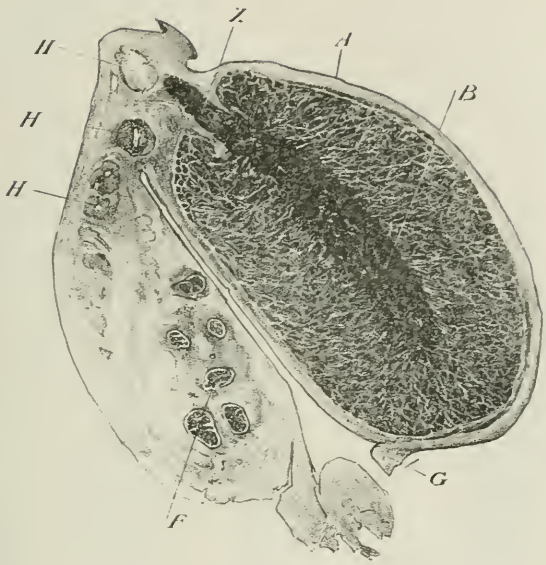



Fig. 10

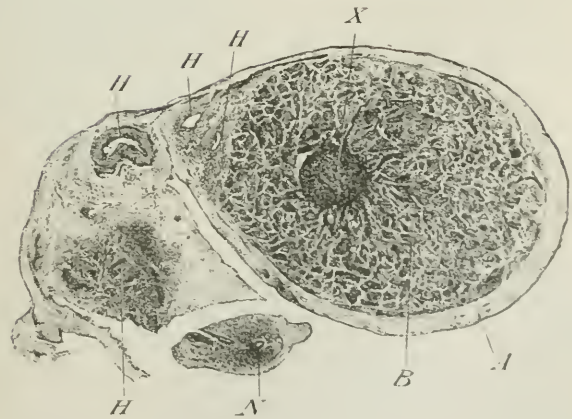



Fig. 11

Fig. 12

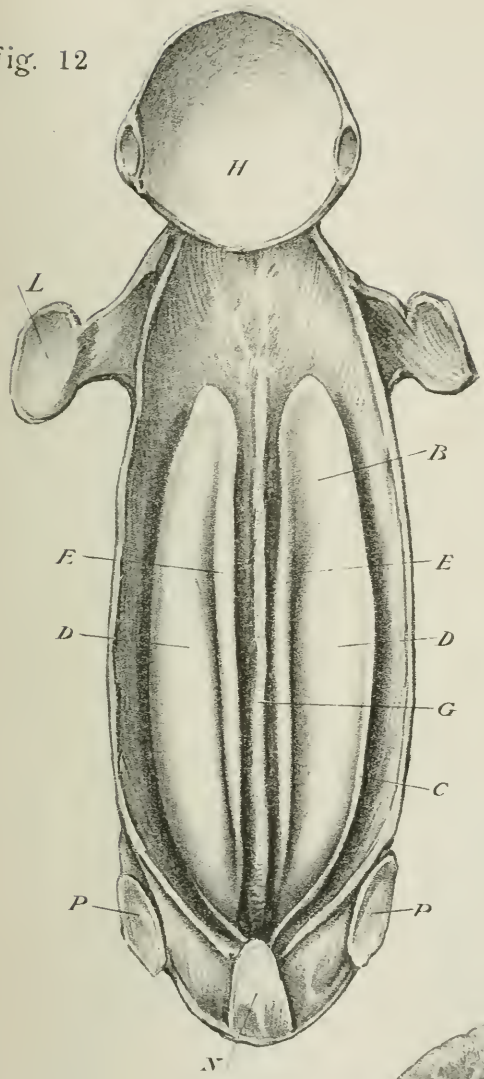



Fig. 13


Fig. 14

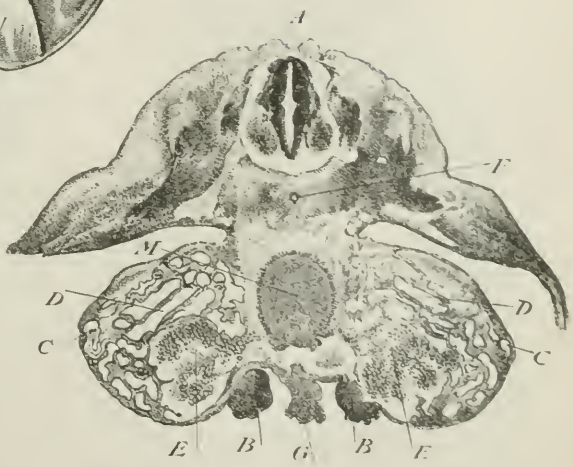


Fig. 15

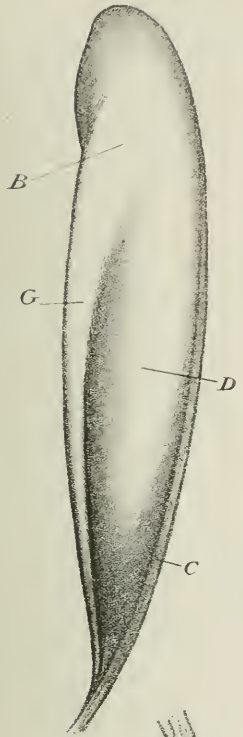
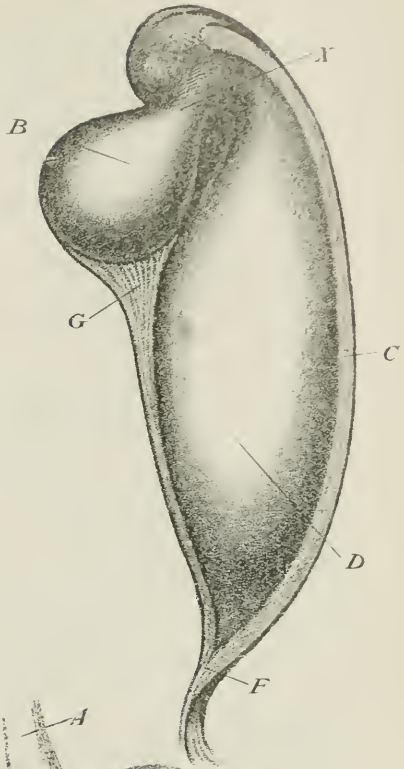



Fig. 16



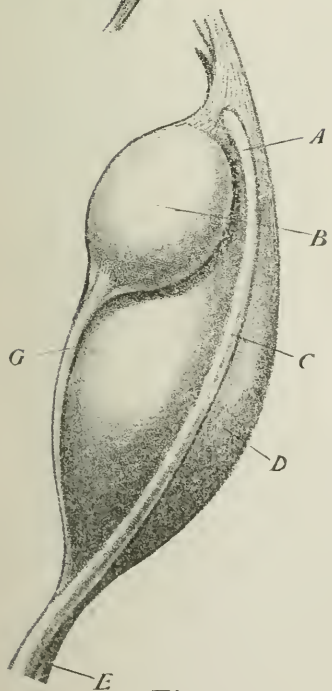




Fig. 17

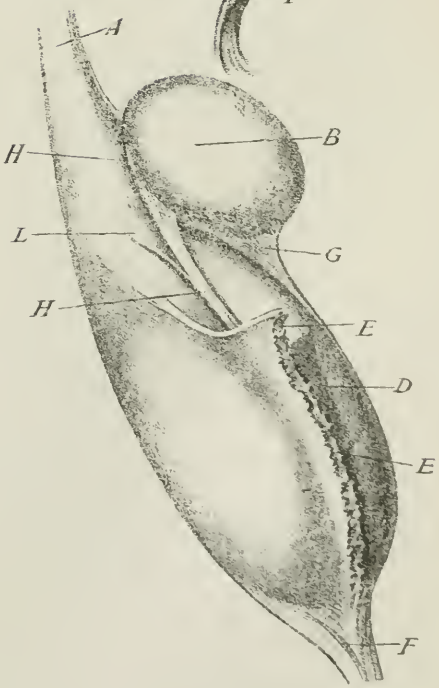



Fig. 18

## Description of Figures.

### *PLATE I.*

Fig. 1.—To show relation of Wolffian Body to Testis and Kidney.

*A* Blind end of Müllerian Duct.
*B* Testicle.
*C* Müllerian Duct.
*D* Wolffian Body.
*E* String formed by Müllerian and Wolffian Ducts.
*G* Gubernaculum Testis.
*H* Junction of Müllerian Ducts in middle line.
*K* Kidney.
*L* Structure, supposed to be the Verum Montanum.
*M* Cut surface of Right Wolffian Body.
*N* Torn surface remaining after the allantoic stalk is removed.

Fig. 2.—The Wolffian Body lying on its side to show the Wolffian Duct passing upwards to Testicle.

*A* Fold of Peritoneum from Wolffian Body and Testicle passing upwards towards diaphragm.
*B* Testicle.
*D* Wolffian Body.
*E E* Torn edge of the Mesorchium.
*F* Wolffian and Müllerian Ducts.
*G* Gubernaculum Testis.
*H H* Wolffian Duct.
*L* Small cyst attached to Wolffian Duct.

### *PLATE II.*

Fig. 3.—The Wolffian Bodies as seen in a fœtal Deer about 2 inches in length.

*A* Fold of Peritoneum passing from upper end of Wolffian Bodies to the diaphragm.
*B* Testis.
*C* Müllerian Ducts.
*D* Wolffian Bodies.
*E* Wolffian and Müllerian Ducts in contact.
*F* Umbilical Artery.
*K* Kidney.
*M* Allantoic stalk.
*R* Rectum.
*X* Stalk of Peritoneum connecting Testicle with Wolffian Body.

Fig. 4.—To show the minute holes in the Capsules of Testicles through which certain tubular structures pass.

*A* Fold of Peritoneum from Wolffian Body.
*B* Hole in Capsule of Testicle.
*C* Müllerian Ducts.
*D* Wolffian Body.
*G* Gubernaculum Testis.
*K* Kidney.
*M* Umbilical Artery.
*N* Allantoic stalk.
*R* Rectum.

### *PLATE III.*

Fig. 5.—Highly magnified view of Wolffian Body to show the hole in the Capsule of Testicle through which certain tubular structures pass.

*A* Folds of Peritoneum passing upwards.
*B* Hole in Capsule of Testicle.
*C* Müllerian Duct.
*F* Müllerian Duct and Wolffian Duct.
*G* Gubernaculum Testis.
*H* Capsule of Testicle, sliced away in part.
*K* Kidney.

### *PLATE IV.*

Fig. 6.—Wolffian Body with Capsule of Testicle attached to show the hole B in Capsule.

*A* Peritoneal Fold passing upwards.
*B* Capsule of Testicle with hole seen at upper part.
*C* Müllerian Duct.
*D* Wolffian Body.
*G* Gubernaculum Testis.

Fig. 7.—Under surface of Wolffian Body to show Wolffian Duct.

*B* Capsule of Testicle.
*C* Wolffian Duct joining Capsule of Testicle.
*D* Wolffian Body.
*E* Cut edge of Mesorchium.
*F* Müllerian and Wolffian Ducts.
*G* Gubernaculum Testis.

Fig. 8.—To show Capsule of Testicle with Wolffian Duct, partly torn away from Wolffian Body.

B Capsule of Testicle with hole in it.
*C* Müllerian Duct.
*D* Wolffian Body.
*G* Gubernaculum Testis.
*H* Stalk from which Capsule of Testicle torn away.
*M* Torn surface on Wolffian Body—the site of the Wolffian Duct, now torn away.

FIG. 9.—Under surface of Wolffian Body to show Capsule of Testicle with Wolffian Duct torn away from stalk and from Wolffian Body.

*B* Capsule of Testicle.
*C* Müllerian Duct.
*D* Wolffian Body.
*H* Stalk of Peritoneum from which Capsule of Testicle is torn.
*L* Torn surface of Capsule of Testicle.
*M* Site of Wolffian Duct, now torn away from Wolffian Body, along with Capsule of Testicle.

*PLATE V.*

FIG. 10.—Vertical section through Wolffian Body and Testis to show origin of Tubuli Seminiferi.

*A* Fibrous Capsule of Testicle continuous with peritoneal stalk.
B Central cellular column from which proceed all the Tubuli Seminiferi.
*F* Glomeruli in Wolffian Body.
*G* Gubernaculum Testis.
*H H H* Cut tubules from Wolffian Duct.
*X* Cellular column passing into Capsule of Testicle to give origin to the Tubuli Seminiferi. This column passes through the centre of the peritoneal stalk.

FIG. 11.—Transverse section of Testicle and Wolffian Body.

*A* Fibrous Capsules of Testicle.
B Tubuli Seminiferi.
*H H H* Cut Tubules of Wolffian Duct.
*X* Transverse section of central cellular column from which arise the Tubuli Seminiferi, in the centre of Testicle.

*PLATE VI.*

FIG. 12.—The Body of an embryo Deer less than one inch in length to show the Wolffian Bodies with the primitive germinal streaks.

*B* Wolffian Body at upper end.
*C* Müllerian Duct.
*D* Wolffian Body.
*E* Primitive germinal streak.
*G* Cut mesentery in middle line.
*H* Head of Embryo.
*L* Anterior limb.
*N* Tail.
*P* Posterior limb.

FIG. 13.—More highly magnified view of one Wolffian Body from this embryo Deer, to show primitive germinal ridge or streak, at its upper part fading off into the Wolffian Body.

*B* Upper part of Wolffian Body with germinal ridge.
*C* Müllerian Duct.
*D* Wolffian Body.
*E* Germinal ridge or streak.

FIG. 14.—Microphotograph—Transverse section through body of this embryo Deer, to show the Wolffian Bodies in section and the young genital glands.

*A* Neural canal.
*B* Young genital gland—Ovary or Testis.
*C* Müllerian Duct in section.
*D* Tubules in Wolffian Body.
*E* Glomeruli with branches from Aorta.
*F* Chorda Dorsalis.
*G* Central Mesentery.
*M* Aorta full of blood corpuscles.

*PLATE VII.*

FIG. 15.—The four figures on this plate are to show the different steps in the development of the Testicle.

*B* The germinal ridge or streak fading off into the Wolffian Body. It is at this spot that the Testicle first appears.
*C* Müllerian Duct.
*D* Wolffian Body.
*G* Primitive germinal ridge or streak.

FIG. 16.—A stage in the development of the Testicle which now appears as an elongated body connected with the Wolffian Body by means of a thick peritoneal stalk.

*B* Testis.
*C* Müllerian Duct.
*D* Wolffian Body.
*F* Müllerian and Wolffian Ducts.
*G* Gubernaculum Testis.
*X* Stalk from the Wolffian Body connected with the out-growing Testicle.

FIG. 17.—A further stage in the development of the Testicle, the stalk is now hidden from view by the larger and more globular Testicle.

B Testicle.
*C* Müllerian Duct.
*D* Wolffian Body.
*E* Müllerian and Wolffian Ducts.
*G* Gubernaculum Testis—the remains of the germinal ridge.

FIG. 18.—Under surface of Wolffian Body to show Wolffian Duct in its relation to the Testicle.

B Testicle.
*E E* Cut Mesorchium.
*H H* Wolffian Duct—The Capsule of Testicle is continuous with the covering of the Wolffian Duct.
*G* Gubernaculum Testis,—which in the female is known as the round ligament of the ovary.

# INDEX

TURNBULL AND SPEARS, PRINTERS, EDINBURGH